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THE ACTION OF MAGNESIUM ON THE HEART

BY

P. SZEKELY

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Received May 12, 1945

Although it has been repeatedly established both in man and in animal experiments that magnesium has a depressant action on the heart, there is no general agreement about the mechanism of this action, and there are few reports dealing directly with the clinical application of its cardio-inhibitory properties. Zwillinger (1935) studied the effect of magnesium sulphate on paroxysmal tachycardia, auricular fibrillation, flutter, and extrasystolic arrhythmia; the paroxysms of tachycardia stopped immediately after the intravenous administration of the drug; extrasystoles were usually abolished; no effect, however, was noticed in auricular fibrillation and flutter. Rothberger and Zwillinger (1936) showed experimentally that magnesium salts in large doses depress conduction, and depress or abolish abnormal impulse production: they succeeded in combating ventricular tachycardia produced by barium chloride or strophanthin. Miller and van Dellen (1938) conducted cardiographic studies in dogs that had been given magnesium sulphate intravenously, and observed a delay in A-V and intraventricular conduction time. Smith, Winkler, and Hoff (1939) reported a sequence of cardiographic changes in the unanæsthetized dog during continuous intravenous administration of magnesium sulphate. They showed that intra-cardiac conduction was depressed in all its elements. After a transient initial tachycardia, bradycardia appeared, and there was a progressive increase in the P–R interval as the concentration increased, until A-V block of various grades and widening of QRS appeared. If the concentration was raised sufficiently, cardiac arrest resulted. Except for the initial tachycardia, similar results were obtained in cats. In the authors' own words—" the cardiac arrest which always occurs if the magnesium concentration is pushed high enough cannot be explained as the result of this depression of conduction, since in most instances the last beats showed a well defined and vigorous systole. For an adequate explanation of this final arrest it is necessary to assume a more direct toxic action of magnesium on the myocardium itself." Bernstein and Simkin (1939) observed only minor changes in the human cardiogram following the intravenous injection of 10 to 20 c.c. of a 10 per cent solution of magnesium sulphate in both cardiac and non-cardiac subjects. These changes consisted of variations in the amplitude of the QRS and T waves. There were no changes in the heart rate or the P–R interval. Boyd and Scherf (1943) recommended magnesium sulphate as a useful therapeutic procedure in paroxysmal tachycardia; using 20 c.c. of a 20 per cent solution they succeeded in terminating each of eight attacks. In one of the paroxysms reported by Decherd and Herrmann (1944) magnesium sulphate, injected intravenously to measure the circulation time, stopped the tachycardia immediately. In a recent report Pines *et al.* (1944) produced further evidence of the cardio-inhibitory property of magnesium sulphate, and showed that the addition of magnesium sulphate to the intravenous or intracardiac injection of a lethal dose of mercurial diuretic prevents ventricular fibrillation and death of the dog.

Present Investigation

The 26 cases included in this study were in-patients of the Newcastle General Hospital under the care of Professor W. E. Hume: they comprise 13 with paroxysmal tachycardia, 9 with auricular fibrillation, 5 of which also showed extrasystoles, and 4 with purely extra-

systolic arrhythmia. Three of the cases of paroxysmal tachycardia might have been regarded as instances of auricular flutter, but they conform to the definition of paroxysmal tachycardia given recently by Evans (1944). Some of the cases of paroxysmal tachycardia and auricular fibrillation were included in the material of a previous study (Szekely, 1944), but none of the cases analyzed here in detail and none of the cardiograms have been reported before. Ten cases with sinus rhythm were also studied with special reference to the effect of magnesium on the rate, the P–R, QRS, and Q–T intervals. In each instance magnesium sulphate was given intravenously, the dose being 20 c.c. of a 20 per cent solution unless stated otherwise.

Case 1. A woman, aged 67, was admitted to hospital in October, 1942, with congestive heart failure and auricular fibrillation. Treatment with digitalis was started at once (digitaline Nativelle, 1/600 of a grain, six times daily). Four days later, she was found to have paroxysmal tachycardia at a rate of 168 a minute (Fig. 1). Digitalis was discontinued and 10 c.c. of a 20 per cent solution of

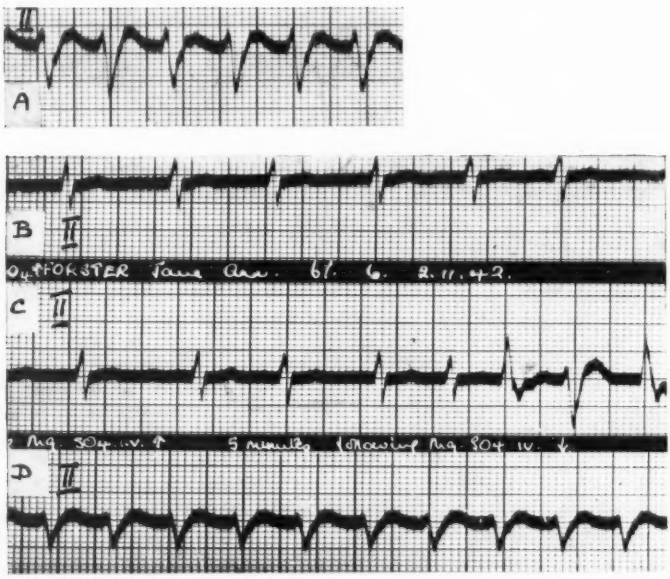

FIG. 1.—Case 1. Paroxysmal tachycardia occurring during auricular fibrillation. (A) Before, (B) Immediately after, (C) 3 minutes after, and (D) 5 minutes after injection of magnesium sulphate. This and subsequent figures have been reduced to five-sixths.

magnesium sulphate were given intravenously. The tracings taken immediately after, and three and five minutes after the injection are reproduced in Fig. 1; they show immediate cessation of the tachycardia and restoration of auricular fibrillation. After three minutes extrasystoles appeared and paroxysmal tachycardia recurred. A second injection of magnesium sulphate, ten minutes later, had the same effect, but this time the tachycardia was arrested for about seven minutes. As the tachycardia recurred and persisted, quinidine sulphate was started by mouth. After three doses each of 5 grains at two-hourly intervals, auricular fibrillation reappeared. Quinidine was then continued, 3 grains daily. Two days later, digitalis was restarted and after two doses of 1/600 of a grain of digitaline Nativelle a rapid regular rhythm developed, presumably paroxysmal tachycardia as before, and the patient died shortly after. Autopsy revealed a moderate degree of diffuse coronary sclerosis with some myocardial fibrosis.

Case 2. A man, aged 59, entered hospital with hypertensive heart failure. He showed the Wolff-Parkinson-White syndrome. The attacks of tachycardia usually lasted for more than twenty-four hours. Intravenous magnesium sulphate terminated one of the paroxysms within five minutes (Fig. 2). As the effect of magnesium is usually immediate (Boyd and Scherf, 1943), it is doubtful whether the disappearance of the tachycardia in this case can be attributed to the drug.

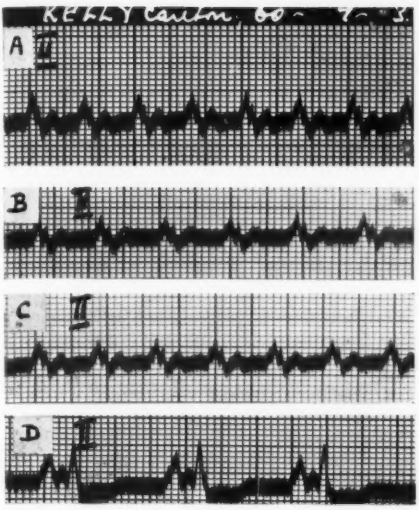

FIG. 2.—Case 2. Paroxysmal tachycardia in case showing Wolff-Parkinson-White syndrome. (A) Before, (B) Immediately after, (C) 2 minutes after magnesium sulphate; no change, and (D) 5 minutes after, showing return of original rhythm.

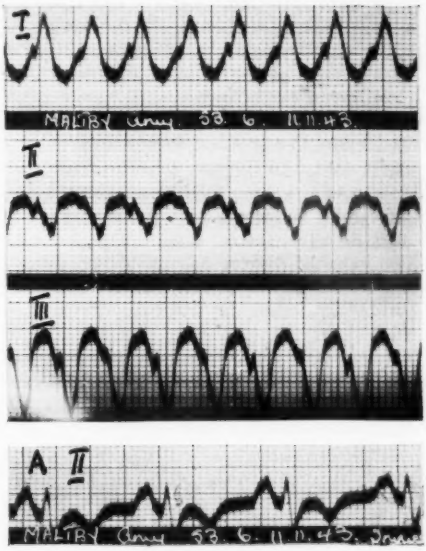

FIG. 3.—Case 3. Mitral stenosis and hypertension. Above, limb leads showing paroxysmal tachycardia, rate 216 a minute. (A) Immediately after magnesium sulphate; sinus rhythm restored.

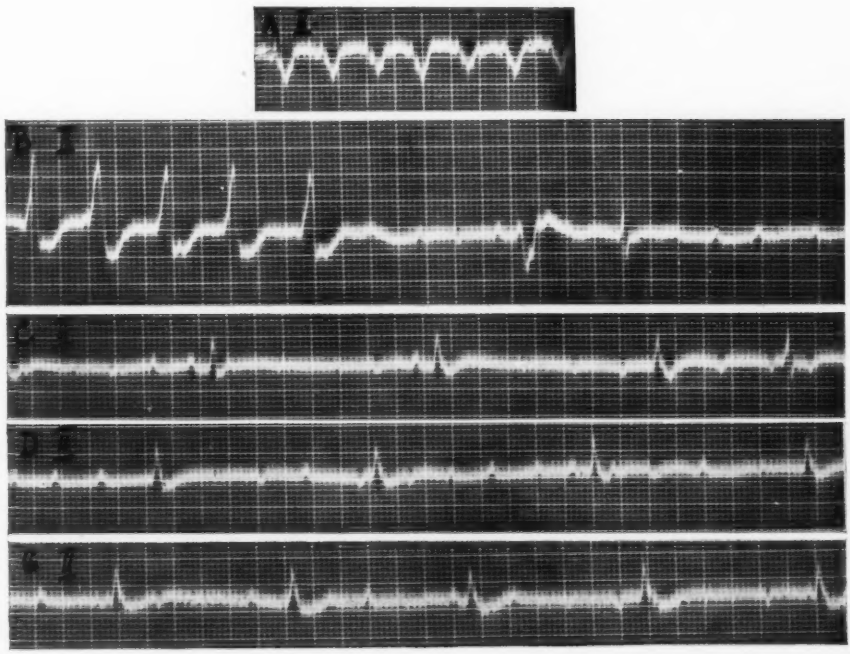

FIG. 4.—Case 4. (A) Before magnesium injection, shows paroxysmal tachycardia, rate 186 a minute. (B), (C), and (D) Continuous strips of lead II after injection. (G) 12 seconds after (D); described in text.

Case 3. A woman, aged 53, was admitted complaining of palpitation. She had taken digitalis for a week before admission. The heart rate was rapid and regular, due to paroxysmal tachycardia, probably ventricular in origin, rate 216 a minute (Fig. 3). The paroxysm was terminated by an intravenous injection of magnesium sulphate and sinus rhythm was restored (Fig. 3). In this case, the effect of magnesium was permanent, and the tachycardia did not recur. During normal rhythm the diagnosis of mitral stenosis and hypertension was established. The patient was discharged two weeks later in a satisfactory condition.

Case 4. A man, aged 74, was admitted with hypertensive heart failure. He had a regular rhythm, rate about 100 a minute. As he did not respond to rest and mercurial diuretics, treatment with digitalis was started. After six days he developed a rapid, regular paroxysmal tachycardia, rate 186 a minute (Fig. 4, A), and digitalis was discontinued. The intravenous injection of 10 c.c. of a 20 per cent solution of magnesium sulphate was followed by a transient change in the shape of the ventricular complexes, but there was no appreciable change in the rate. A few hours later, the same dose of magnesium sulphate was repeated and resulted in immediate changes (Fig. 4, B, C, D, G). After a short run of ventricular tachycardia, rate about 125 a minute, complete A-V dissociation appeared—A., 210; V., 38 gradually increasing to 50. There was variation in the shape of the auricular complexes and alternation of the cycle length. The slow ventricular rate persisted for about eighteen hours, then again rose to about 180 a minute. The patient died shortly after. No autopsy was done.

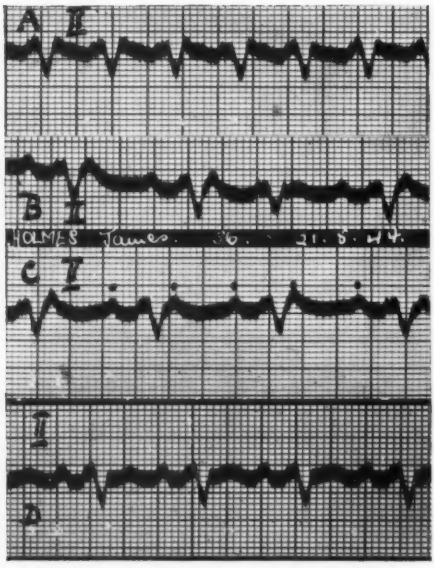

FIG. 5.—Case 5. (A) Paroxysmal tachycardia and probably 2 : 1 A-V block. (B) Immediately after injection of magnesium; 2 : 1 A-V block, auricular rate 170. (C) 3 minutes after; no change. (D) 20 minutes after; sinus rhythm with original bundle branch block pattern.

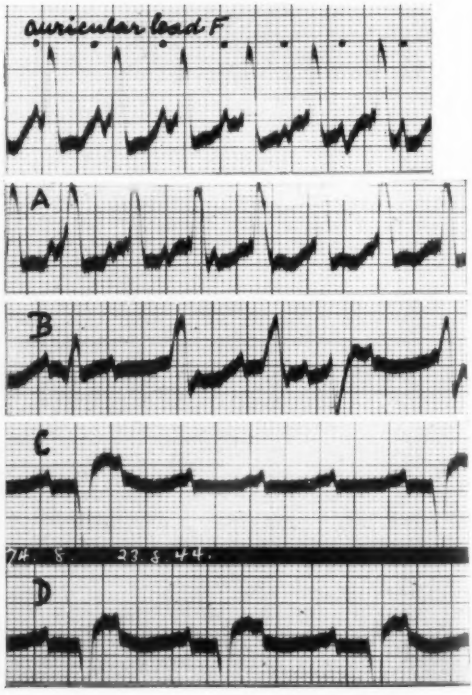

FIG. 6.—Case 6. Auricular chest lead. Upper tracing. Paroxysmal tachycardia with A-V block, dots denote P waves; auricular rate 170, ventricular rate slightly slower. (A) During injection of magnesium; auricular rate 186. (B) Immediately after; auricular rate 170, multifocal ventricular beats. (C) 2 minutes after; auricular rate 150, ventricular rate 30. (D) 3 minutes after; auricular rate 150, ventricular rate 75.

Case 5. A man, aged 56, was admitted to hospital on April 28, 1944, with hypertensive heart failure. He had a large heart, triple rhythm, and left bundle branch block. Treated with digitalis and mercurial diuretics, he improved, but on May 25 his heart rate increased suddenly—paroxysmal tachycardia with 2 to 1 A-V dissociation; auricular rate 340 a minute (Fig. 5, A). Digitalis was discontinued, and the magnesium sulphate was given intravenously. The tracings taken immediately and at intervals of three and five minutes after the injection showed 2 : 1 A-V block, auricular rate about 170 a minute (Fig. 5, B, C). The tracing in Fig. 5, D, taken twenty minutes after the injection, shows sinus rhythm and the original bundle branch block pattern. There was no recurrence of the tachycardia and the patient was discharged ten days later in a satisfactory condition.

Case 6. A man, aged 74, entered the hospital with congestive heart failure. He had an enlarged heart with normal blood pressure and regular rhythm. Four days after admission the heart rate suddenly increased and Fig. 6 showed paroxysmal tachycardia with A-V dissociation, which is clearly shown in the auricular lead. The auricular rate was 170 a minute and the ventricular rate slightly slower. The intravenous injection of magnesium sulphate resulted in only slight changes in the auricular rate, but greater changes in the ventricular rate (Fig. 6, A, B, C, D). After a slight initial rise the auricular rate fell to 150 a minute. The ventricular rate fell to about 30 a minute and then rose to 75 a minute, this rate persisting until the death of the patient three days later. Unfortunately no record was taken before the onset of the tachycardia, so that it is impossible to say what the original rhythm was.

Case 7. A man, aged 23, was first seen in April 1942, and again in November 1944, with mitral stenosis and congestive failure, with normal rhythm. Shortly after admission, paroxysmal tachycardia was recorded, ventricular rate 176 (Fig. 7). Within a few minutes, the deflection of the string changed and lead III, recorded five minutes after the first curve, showed an increased widening of QRS without any change in rate (Fig. 7, A). Magnesium sulphate was then given intravenously ; a curve taken immediately showed no change (Fig. 7, B); after 1½ minutes the QRS returned to its original form (Fig. 7, C); after 2½ minutes sinus rhythm was recorded (Fig. 7, D), and there was no recurrence of tachycardia up to the patient's death six days later. No autopsy was obtained.

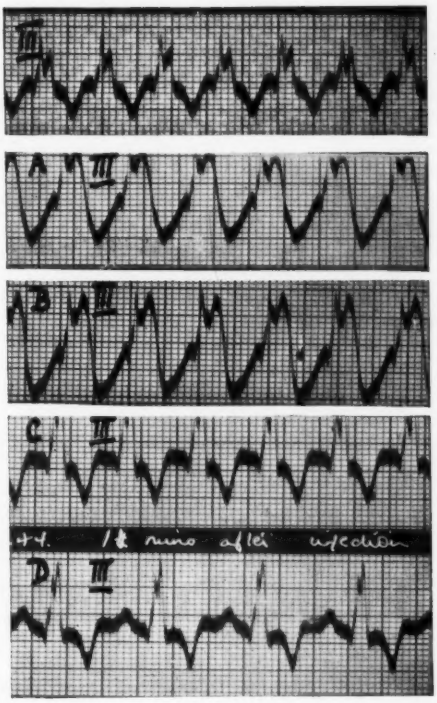

FIG. 7.—Case 7. Lead III. Upper tracing ; paroxysmal tachycardia, ventricular rate 176 ; probably 2 : 1 A-V block. (A) Spontaneous change of QRS. (B) Immediately after magnesium ; no change. (C) 1½ minutes after ; QRS returned to original form. (D) 2½ minutes after; sinus rhythm restored.

Case 8. A man, aged 39, had coronary occlusion with hemiplegia and *Streptococcus viridans* septicæmia. His initial cardiogram showed changes characteristic of anterior myocardial infarction. He died two months after admission and the diagnosis was confirmed at autopsy. Five days prior to death the rhythm changed to auricular fibrillation. About four hours later, a regular tachycardia, auricular rate 500 a minute, was recorded (Fig. 8). Intravenous injection of magnesium sulphate slowed the auricular rate to 300 and the ventricular rate to 150 a minute (Fig. 8, A, B, C). The original rate recurred the following day and persisted until the patient's death.

Case 9. A woman, aged 62, was admitted in 1944, with hypertensive heart failure and left hemiplegia. The cardiogram showed paroxysmal tachycardia with 2: 1 A-V block (Fig. 9, A), auricular rate 320 a minute. Magnesium sulphate intravenously increased the A-V block (Fig. 9, C); 4 : 1 A-V block persisted for an hour, when the original 2: 1 block reappeared and persisted until death three days later. Autopsy refused.

Case 10. A woman, aged 51, had been seen for several years with rheumatic heart disease and auricular fibrillation. She died in hospital and autopsy confirmed the diagnosis. During her last three years she had practically never been without digitalis. On one occasion extrasystolic bigeminy was recorded (Fig. 10, A). After an injection of magnesium sulphate the extrasystoles were not completely abolished but their voltage was reduced (Fig. 10, B and C); the voltage of the normal ventricular complex was unaffected. This demonstrates the depressant action of magnesium on ectopic centres, while the normal complexes remain unchanged. The duration of this effect was not observed.

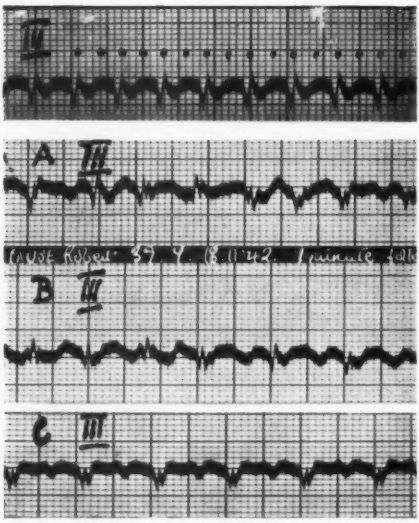

FIG. 8.—Case 8. Coronary occlusion. Upper tracing. Auricular tachycardia with 2 : 1 block, auricular rate 500. (A) 1 minute after magnesium ; variable A-V block. (B) 2 minutes after ; auricular rate 300, variable A-V block. (C) 4 minutes after; auricular rate 300, 2 : 1 A-V block.

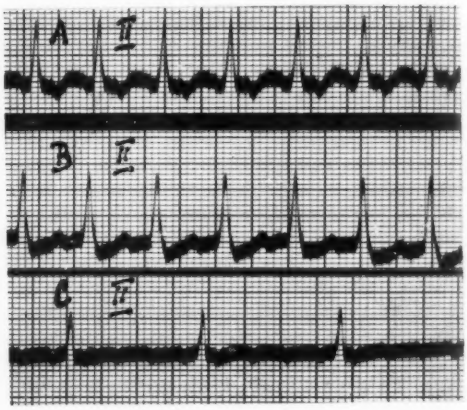

FIG. 9.—Case 9. (A) Auricular tachycardia with 2 : 1 block, auricular rate 320. (B) During injection of magnesium ; change in shape of auricular waves. (C) Immediately after ; ventricular rate halved.

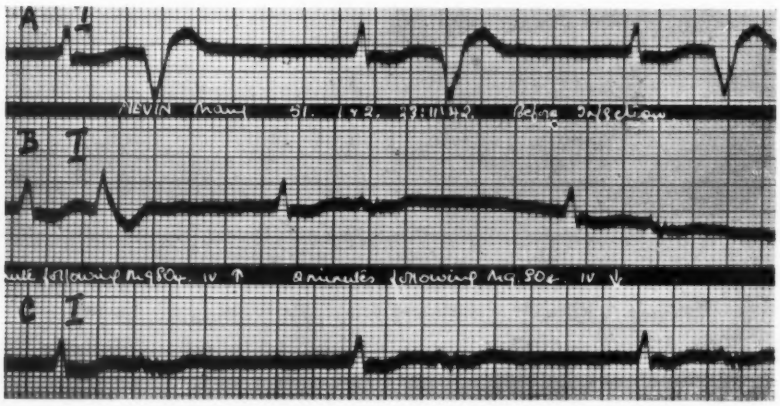

FIG. 10.—Case 10. (A) Auricular fibrillation and extrasystolic bigeminy in a digitalized patient. (B) and (C) 1 and 2 minutes respectively, after injection of magnesium. Decrease in voltage of extrasystolic beat. Voltage of normal complex remains unchanged.

Case 11. A woman, aged 77, was admitted in 1944 with congestive heart failure. The rhythm was regular. Though there was no history of cardiac pain, the low blood pressure and the chest lead cardiogram suggested anterior myocardial infarction. She was given digitalis and mercurial diuretics, and improved; after three weeks treatment extrasystolic bigeminy and 2 : 1 A-V block was recorded (Fig. 11). After an intravenous injection of magnesium sulphate the extrasystoles were completely abolished, and did not recur; the patient was discharged ten days later in a satisfactory condition.

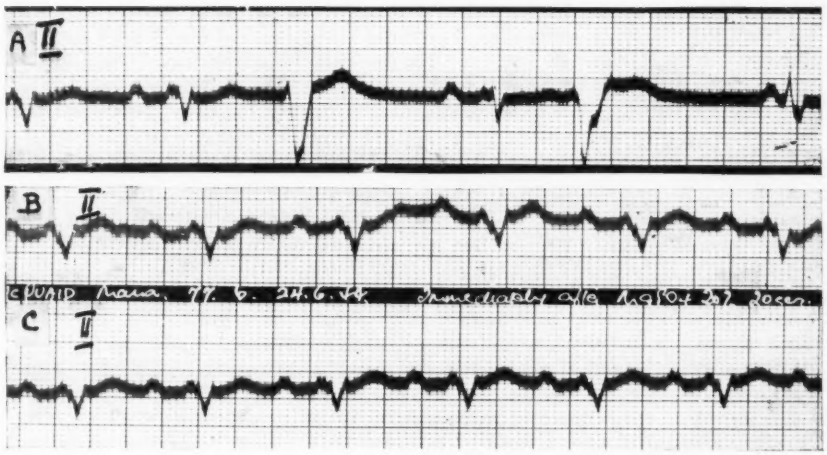

FIG. 11.—Case 11. (A) Extrasystolic bigeminy and 2 : 1 A-V block due to digitalis. (B) Immediately after magnesium and (C) 2 minutes after : sinus rhythm with prolonged P–R ; extrasystoles abolished.

DISCUSSION

The findings confirm the views of previous investigators that magnesium has a depressant action on the heart muscle. The results obtained in paroxysmal tachycardia are summarized in Table I. Of 13 cases, 9 were affected by magnesium, and in 5 of them (Cases 2, 3, 5, 6, and 7) the effect appeared to be lasting; in 4 of them sinus rhythm was restored, and in the fifth case, though the ventricular rate was much reduced, the auricular rate was only slightly slowed, and the A-V dissociation, originally present, persisted. In 4 (Cases 1, 4, 8,

TABLE I

THE EFFECT OF MAGNESIUM ON PAROXYSMAL TACHYCARDIA

Case Number	Underlying heart disease	Auricular rate	Ventricular rate	Duration of effect	Rhythm after injection of magnesium
1 (Fig. 1)	Coronary sclerosis	?	Decreased	3 and 7 minutes	Auricular fibrillation
2 (Fig. 2)	Hypertension	Decreased	Decreased	Lasting	Sinus rhythm
3 (Fig. 3)	Mitral stenosis and hypertension	Decreased	Decreased	Lasting	Sinus rhythm
4 (Fig. 4)	Hypertension	Increased ?	Decreased	18 hours	A-V block
5 (Fig. 5)	Hypertension	Decreased	Decreased	Lasting	Sinus rhythm
6 (Fig. 6)	Coronary sclerosis	Decreased	Decreased	Lasting	A-V block
7 (Fig. 7)	Mitral stenosis	Decreased	Decreased	Lasting	Sinus rhythm
8 (Fig. 8)	Coronary thrombosis	Decreased	Decreased	24 hours	2 : 1 A-V block
9 (Fig. 9)	Hypertension	Unchanged	Decreased	1 hour	4 : 1 A-V block
12	Nil	Unchanged	Unchanged	—	Unchanged
13	Nil	Unchanged	Unchanged	—	Unchanged
14	Mitral and aortic disease	Unchanged	Unchanged	—	Unchanged
15	Nil	Unchanged	Unchanged	—	Unchanged

and 9) the effect was transient, ranging from three minutes to twenty-four hours. In Case 1 the basic rhythm was auricular fibrillation, and this recurred both times the tachycardia was terminated by magnesium. In Case 4, A-V dissociation was present before magnesium was given; after the injection, the auricular rate appeared to be quicker, and there was a marked alternation of the cycle length; the ventricular rate showed a fall. In Cases 8 and 9 the injection resulted in a temporary increase in the A-V block. Magnesium failed to produce any changes in rhythm in the following 4 cases. A male, aged 63, with nodal tachycardia and no underlying structural disease in the heart, whose tachycardia responded to carotid sinus pressure. A male, aged 36, with auricular tachycardia but no structural disease of the heart, whose tachycardia was terminated by digitalis. A female, aged 17, with mitral stenosis and aortic regurgitation, but free from cardiac enlargement or failure; paroxysmal auricular tachycardia stopped after oral digitalization. A male, aged 50, who had auricular tachycardia with 2:1 A-V block, emphysema, and chronic bronchitis; digitalis first resulted in an increase of the A-V block, and later the tachycardia was replaced by sinus rhythm. Clinically, the interesting feature was the presence of advanced heart disease usually with congestive heart failure in the cases that responded to magnesium, even if the effect was a temporary one, and the absence of these findings in the cases that failed to respond to magnesium. Three of the latter showed no evidence of structural heart disease, and in the fourth there were no signs of heart failure though the heart showed structural changes. The significance of this observation is uncertain as yet, but the question may be usefully discussed in relation to the cardiac action of potassium. Though there is some evidence that potassium may be antagonistic to magnesium (Hirschfelder, 1929; Rothberger and Zwillinger, 1936), there are numerous reports (Ringer, 1882; Sampson and Anderson, 1930; Wiggers, 1930; Winkler, Hoff, and Smith, 1938; Thomson, 1939; Stewart and Smith, 1941; Keith, Osterberg, and Burchell, 1942; Finch and Marchand, 1943; Sampson, Alberton, and Kondo, 1943; Keith, Burchell, and Baggenstoss, 1944) suggesting that the cardio-inhibitory action of potassium is in many respects strikingly similar to that of magnesium. Further, it has been shown that the potassium content of the heart muscle of patients dying of congestive heart failure is diminished (Harrison, Pilcher, and Ewing, 1930; Sampson and Anderson, 1930).

Potassium salts have been used to abolish paroxysmal tachycardia and ectopic beats (Sampson and Anderson, 1930), and it has been suggested that potassium is more effective in this respect when the heart muscle is damaged than when it is healthy. This is ascribed to the low potassium content of the myocardium in heart failure. It is possible that the effect of magnesium in paroxysmal tachycardia depends similarly on the magnesium content of the heart muscle.

Sampson and Anderson (1930, 1932) and Sampson, Alberton, and Kondo (1943) showed that ectopic beats caused by digitalis could always be abolished by potassium, whereas other forms of ectopic beat responded less frequently. There is a loss of potassium from the heart muscle in digitalis intoxication (Sampson, Alberton, and Kondo, 1943). Like potassium, magnesium may be effective in digitalis intoxication (Zwillinger, 1935), and it may be more effective in abolishing ectopic beats due to digitalis than those otherwise caused (Zwillinger, 1935; Herles, 1937; Boyd and Scherf, 1943). The findings given in Table II suggest that magnesium is more likely to affect extrasystoles due to digitalis than other varieties. However, reports dealing with the relation between magnesium and digitalis are contradictory, for Miller and van Dellen (1941) found that magnesium did not counteract digitalis intoxication in dogs. Neither potassium nor magnesium has any effect on the rhythm in auricular fibrillation.

The action of magnesium on the heart has been explained as due to a depression of the cardiac nervous mechanism resulting in loss of tone (Matthews and Brooks, 1910), to a vagal effect, and to a direct effect on conduction (van Dellen and Miller, 1939). Smith, Winkler, and Hoff (1939) maintain that magnesium depresses the S-A, A-V, and intraventricular conduction. Pines *et al.* (1944) observed disturbances of A-V and intraventricular conduction from large doses of magnesium, but with smaller doses they were able to abolish ventricular fibrillation due to mercurial diuretics without affecting conduction. These findings suggest that magnesium depresses ectopic centres more than the normal conducting system, but that both are depressed by large enough doses.

To gain more information on the action of magnesium upon the conducting system, 10 cases with sinus rhythm were studied. In 5 of them the cardiogram was normal, and in 5 it showed T wave changes. In each case the limb leads were first recorded, and then usually lead II, immediately after the injection of 20 c.c. of a 20 per cent solution of magnesium sulphate, and two minutes later. Besides minor alterations in the rate in 4 cases, and the appearance of ventricular extrasystoles in 1, no changes were observed. These results are in agreement with those of Bernstein and Simkin (1939) who studied the effect of magnesium on the human

TABLE II

THE EFFECT OF MAGNESIUM ON EXTRASYSTOLIC ARRHYTHMIA

Case Number	Underlying heart disease	Cause of extra-systoles	Effect	Duration of effect
10 (Fig. 10)	Mitral stenosis	Digitalis	Decrease in voltage	Unknown
11 (Fig. 11)	Coronary thrombosis	Digitalis	Extrasystoles abolished	Lasting
16	Hypertension	Digitalis	Extrasystoles abolished	Ten minutes
17	Mitral stenosis	Digitalis	Change in shape of extra-systoles	Unknown
18	Mitral stenosis	Digitalis	Trigeminy	Few minutes
19	Mitral stenosis	Spontaneous	Nil	—
20	Nil	Spontaneous	Nil	—
21	Coronary thrombosis	Spontaneous	Nil	—
22	Mitral stenosis	Spontaneous	Nil	—

heart in normal cases and in those suffering from various forms of heart disease, and found that magnesium had no effect on the normal conductive system. The appearance of extrasystoles is a paradoxical effect which has also been noticed by others (Miller and van Dellen, 1941; Boyd and Scherf, 1943).

The intravenous administration of magnesium sulphate, in doses up to 20 c.c. of a 20 per cent solution, is a safe procedure and we have seen no untoward effect from it. It causes an unpleasant but transient feeling of intense heat due to a vasodilator action. Though several patients in this series died, the death was unrelated to magnesium sulphate; it was due in every case to the heart failure and condition of the heart that initiated the attack. The time interval between the giving of magnesium sulphate and death ranged from twenty hours to three months. The normal magnesium content of the serum is 2 to 3 mg. per 100 c.c. According to Goodman and Gilman (1943), the kidneys excrete magnesium so rapidly that the oral ingestion of magnesium salts results in only a slight rise in the serum magnesium. It is to be expected, therefore, that the serum magnesium, following a single intravenous injection of magnesium sulphate, will return very quickly to the original level. However, by continuous intravenous injection, Smith, Winkler, and Hoff (1939) were able to raise considerably the serum magnesium level. It is generally agreed that the excretion of magnesium is decreased, and correspondingly the serum magnesium increased in patients with renal failure (Hirschfelder and Haury, 1934; Brookfield, 1937; Haury, 1942; Winkler, Smith, and Hoff, 1942). Rubin and Rappaport (1941) warned against the possible danger of administering magnesium to anuric patients, and Hirschfelder and Haury (1934) concluded that hyper-magnesæmia was an important causal factor in many cases of so-called uræmic coma. One should be cautious, therefore, in giving magnesium to patients with renal failure.

SUMMARY AND CONCLUSIONS

The effect of intravenous injections of magnesium sulphate, 20 c.c. of a 20 per cent solution on the human electrocardiogram was studied in cases with sinus rhythm, paroxysmal tachycardia, extrasystolic arrhythmia, and auricular fibrillation.

It was found to have no significant effect on the normal conducting system, and it produced only minor changes in the normal cardiogram.

Out of 13 cases of paroxysmal tachycardia 9 were affected by magnesium sulphate; in 5 the effect was lasting, and in 4 only temporary, *i.e.* from three minutes to twenty-four hours. In 4 sinus rhythm was restored, in 1 the tachycardia was replaced by the original

rhythm of auricular fibrillation. In the remaining 4 cases variable degrees of A-V block appeared. A dose of magnesium sulphate that has no appreciable effect on the normal conducting system, may depress impulse production in heterotopic centres. In increased myocardial irritability all parts of the conducting system may be depressed. The data presented justify the use of magnesium sulphate as a therapeutic agent in paroxysmal tachycardia. The intravenous administration of magnesium sulphate is a safe procedure, but its effect may be only temporary.

Extrasystoles caused by full digitalization were either completely abolished or altered by magnesium, while extrasystoles from other causes were not affected by it. Further investigation of the relationship between magnesium and digitalis is required.

Auricular fibrillation was not affected by magnesium.

The conclusions reached apply only to the human heart and to doses of magnesium sulphate specified. The results of animal experiments are not directly comparable.

I am indebted to Professor Hume for his helpful criticism and advice. I also wish to thank Dr. William Evans for his help and suggestions, Dr. I. E. McCracken, Medical Officer of Health, Newcastle-on-Tyne, and Dr. G. Hurrell, Medical Superintendent, Newcastle General Hospital, for facilities provided, and the Editors of this Journal for their help in reducing and rearranging the paper.

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RUPTURED CONGENITAL ANEURYSM OF THE POSTERIOR SINUS OF VALSALVA

BY

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The three sinuses of Valsalva are best described as right, left, and posterior, the last being the non-coronary sinus. An aneurysm may develop in relation to any of these, but the right sinus is the one most usually involved; the aneurysm then generally ruptures into the right ventricle. The posterior sinus is far less often implicated. It comes into close anatomical relationship to the right atrium, into which an aneurysm of this sinus is likely to rupture. Symptoms are unusual until shortly before death, which is often sudden, through perforation into the pericardium, pulmonary artery, right atrium, or ventricle. Those aneurysms that enlarge to the right may cause conduction disturbances by their proximity to the membranous portion of the interventricular septum.

Syphilis, ulcerative endocarditis, and atheroma are recognized causes of dilatation of the aortic sinuses, but the condition also occurs as a rare congenital anomaly. Abbott (1936) was able to collect records of 12 congenital cases; Micks (1940) described a case of congenital aneurysms of all three sinuses; while Hirschboeck (1942) reported an aneurysm of the right sinus, and Macleod (1944) one of the left. Brown (1939) mentioned two cases, in one of which the aneurysm arose from the right sinus and in the other from all three; in both these rupture occurred into the pericardium: he stated that in about half the congenital examples there was an additional defect of the bulbar septum just below the aneurysm.

Report of a Case

A housewife, aged 31, was admitted to Southmead Hospital on February 6, 1946, with symptoms and signs of heart failure.

At the age of 12, because she had had frequent fainting attacks, she was referred to Dr. Coombs at the Bristol Royal Infirmary. On examination he found that the apex beat was normal in position and extent. Pulsation was felt in the epigastrium. A loud musical systolic murmur was heard over the whole of the præcordium, maximal at the inner ends of the fourth and fifth spaces; it could also be heard between the left scapula and the spine. There was no cyanosis and no clubbing of the fingers. An X-ray did not show any cardiac enlargement, and the cardiogram was normal. There was no history to suggest rheumatic fever. Seen again a year later by Dr. Coombs, her condition was found to be unchanged. He considered the heart lesion to be congenital.

The patient herself was adamant that until two weeks before admission to hospital she was able to indulge in all normal activities without symptoms. She had passed through three labours without undue distress, and in none had she to take to her bed before the actual onset of labour pains. She had a vague recollection of having attended a cardiac clinic in youth but understood that she had outgrown her heart trouble.

Two weeks before admission, whilst sawing wood, she experienced a sudden, acute pain behind the lower part of the sternum, which radiated upwards into the throat and gave her a choking sensation. Because of the severity of the pain, and the repeated vomiting which soon accompanied it, she was forced to go to bed. The pain and vomiting continued for the following two days. She became increasingly short of breath and her legs and abdomen began to swell. Insomnia became troublesome.

On admission she was found to be dyspnœic. Her face was pale except for slight cyanosis in the cheeks and lips. There was engorgement of the cervical veins and those coursing over the upper part of the chest; these vessels showed pulsation. The apex beat was in the sixth space, two inches outside the mid-clavicular line. No thrills were felt. The heart was beating regularly at the rate of 120 a minute. All over the præcordium there was a rough, roaring systolic and a diastolic murmur. This was much louder over the tricuspid area than anywhere else, but it was also very loud over the

mitral area. Listening over the aortic area and along the left border of the sternum it was difficult to decide whether the murmurs heard were conducted from the tricuspid area or whether they were different in character and originating from the aortic valve. The blood pressure was 200 systolic, and 40 diastolic. There was dulness over both lung bases with diminished air entry and crepitations, more so on the right. There was a moderate amount of free fluid in the abdomen. The liver was enlarged three fingers' breadth below the costal margin, and could be felt to pulsate. The spleen was not palpable. There was gross œdema of the legs and the small of the back.

An antero-posterior X-ray of the chest, taken with a portable machine, showed gross dilatation of the heart and an opacity at the base of the right lung. A cardiogram revealed a regular A-V nodal rhythm at a rate of 120 a minute, and right ventricular preponderance (Fig. 1). The hæmoglobin

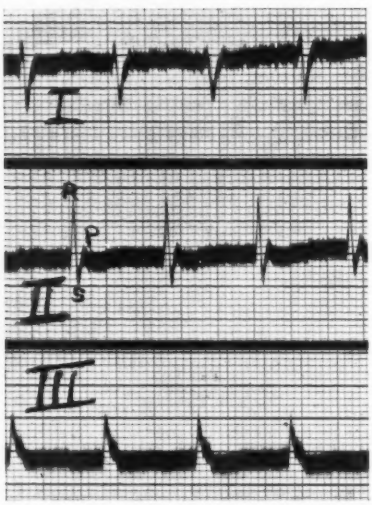

FIG. 1.—A-V nodal rhythm and right axis deviation. The heart is beating regularly at a rate of 120 a minute.

was 78 per cent (Haldane), the red blood corpuscles 4,400,000, and the white blood corpuscles 14,000 per cu. mm. Carotid and eyeball pressure had no effect on the cardiac rate. Treatment with restricted fluid and salt intake, full doses of digoxin, injections of mersalyl, and with sedatives produced no improvement, and had very little effect on the cardiac rate. The œdema and effusions into the serous cavities increased—the ascites had to be tapped. On February 25 her conjunctivæ were slightly jaundiced and urinary output was poor. She died rather suddenly at about noon on this day, shortly after addressing a remark to one of the nurses.

Post-mortem examination was performed 24 hours after death.

The body was well nourished, there was slight jaundice present and generalized venous engorgement with anasarca.

The abdominal and pleural cavities contained a large quantity of bile-stained fluid and the pericardial cavity also contained a slight excess of similar fluid.

The heart weighed 385 g. The right side was considerably dilated; the right atrium communicated with the left through a small aperture in the fossa ovalis; beneath this, lower down the interatrial septum and just above the septal cusp of the tricuspid valve, there was a saccular projection of thin membrane in the centre of which was a small hole 1 mm. in diameter; there was also a ragged perforation of this aneurysmal sac through a calcified part of its wall (Fig. 2). In the anterior margin of the septal cusp of the tricuspid valve there was a foramen 3 mm. in diameter leading directly into the left ventricle. The margins of the valve adjacent to this aperture were thickened and slightly discoloured: this was presumably due to the mechanical stress of the jet of blood entering from the left ventricle. The valve measured 13 cm. in circumference and appeared competent.

The right ventricular wall measured 7 mm. in thickness and the chamber appeared somewhat dilated. The pulmonary valve was normal.

The left atrium was not remarkable.

The mitral valve measured 9 mm. in circumference and was healthy.

The left ventricular wall measured 15 mm. in thickness and was not dilated or hypertrophied. Just below the point of fusion of the right and posterior cusps of the aortic valve there was a foramen opening through the anterior margin of the septal cusp of the tricuspid valve as described above.

The aortic valve was deformed; the right and posterior cusps were partially fused and there was a small fenestration in the free margin of the right cusp. The adjacent parts of these cusps were thickened (Fig. 3), but there was no generalized thickening as seen in rheumatic valvulitis. The valve

FIG. 2.—View of right atrium and tricuspid valve. The arrow points to the ruptured aneurysm with the orifice of the patent interventricular septum immediately below it. Note the roughening of the tricuspid leaflets. Magnification ×1·4.

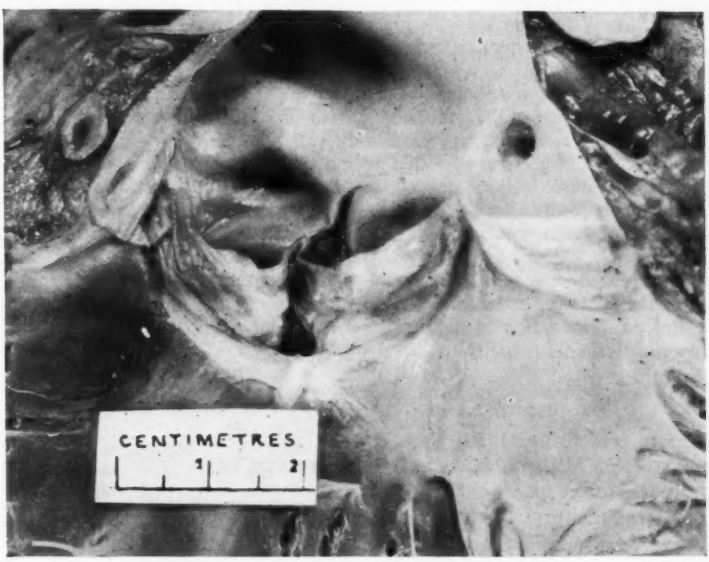

FIG. 3.—Aortic valve. The sinuses of Valsalva enumerated from left to right are: right, posterior, and left. The interventricular septal defect lies immediately below the fused right and posterior cusps. Magnification ×1·4.

FIG. 4.—Sinuses of Valsalva as viewed from above.
(A) Aneurysm of right sinus.
(B) Aneurysm of posterior sinus leading into right atrium.
(C) Normal left sinus. Magnification ×1·4.

measured 6 cm. in circumference (normal 8 cm.). The right sinus of Valsalva was considerably dilated and stretched back into the muscle of the interventricular septum. The floor of the posterior sinus of Valsalva consisted of an aneurysmal sac leading into the right atrium (Fig. 4). The left sinus of Valsalva was normal.

There was no evidence of any rheumatic or infective endocarditis, past or present.

There was slight atheroma of the abdominal aorta. The left renal vein passed behind the aorta.

Both lungs were very congested and moderately œdematous.

The spleen was not enlarged and appeared normal on section.

The liver showed intense venous congestion, and histologically there was considerable central necrosis, sufficient to account for the jaundice. There was no evidence of chronic congestion.

The gall-bladder and bile ducts were healthy and patent.

Both kidneys showed venous congestion. There was a double ureter on the left side.

Anatomical diagnosis. Congenital aneurysms of the right and posterior aortic sinuses of Valsalva with rupture of the posterior sinus into the right atrium. Patent interventricular septum. Fusion of the right and posterior cusps of the aortic valve. Right ventricular hypertrophy. Congestive cardiac failure with terminal hepatic jaundice.

DISCUSSION

Some writers (Goehring, 1920) have considered a deformity of the aortic valve cusps similar to ours to be a bicuspid valve, but this is not admissible as the adjacent raphé between the right and posterior cusps was still present.

Histological examination of the liver showed that the venous congestion was of recent origin, and it seems probable therefore that it dated approximately from the time of rupture of the aneurysm.

The sudden onset with pain, shock, and vomiting, signs of aortic insufficiency, and the rapid development of heart failure in a previously healthy subject characterize most of the

detailed reports of this condition. This clinical picture may also be produced by rupture of an aortic valve cusp and sometimes by a dissecting aneurysm involving the aortic ring. As in coronary occlusion, leucocytosis and even elevation of the temperature may be found. In both the case reported by Micks (1940) and in that by Duras (1944) there was heart block. Other conduction disturbances have been recorded.

In many of the reports the clinical details are scanty, because of the rapidity with which death follows rupture. Both Hirschboeck (1942) and Macleod (1944) reported rupture of an aortic sinus into the right atrium; in both these cases and in our own the clinical pictures were very similar.

In the case reported by Hirschboeck the aneurysm arose from the right sinus of Valsalva. There was a systolic thrill over the apex, and a harsh systolic murmur was audible at the lower end of the sternum and in the epigastrium. On admission the blood pressure was 300/0 and the heart rate was 140 a minute. Liver dulness extended about two fingers' breadth below the costal margin. The leucocytes were counted on two occasions—on the first occasion the count was 11,400 per cu. mm. and on the second 18,800 per cu. mm. The temperature fluctuated from normal to 103° F. Five days after admission a diastolic murmur was heard at the base and along the left border of the sternum. A diagnosis of aortic and tricuspid insufficiency caused by endocarditis of undetermined origin was made. Right axis deviation had been found in the cardiogram and in view of the extreme hypertension this was considered to be unusual. Macleod described the onset of symptoms in his patient as sudden with abdominal pain, vomiting, and breathlessness. A harsh systolic murmur was heard all over the heart, loudest in the aortic area, and at the right border of the sternum there was a soft diastolic murmur. There was a water-hammer pulse, and engorgement and pulsation of the veins of the neck. A cardiogram showed auricular fibrillation. Necropsy, five weeks after onset, revealed an aneurysm of the left sinus of Valsalva, which had ruptured into the right atrium.

In our case the murmur noted in childhood was no doubt due to the communication between the left ventricle and the right atrium. This lesion does not appear to have caused any gross functional disturbance.

The sudden onset of symptoms can best be explained by rupture of the posterior sinus of Valsalva into the right atrium. The loading of the right atrium with blood from the aorta during auricular diastole explains the venous congestion and the pulsation in the veins of the neck and liver, while the same leak of blood out of the aorta accounts for the marked water-hammer pulse. The roaring systolic and diastolic murmur, heard best over the tricuspid area, is also accounted for by the anatomical findings. From a functional point of view a similar disturbance of cardiac dynamics would result from a combination of tricuspid and aortic regurgitation, and this was the diagnosis during life.

The A-V nodal rhythm was probably due to irritation of the A-V node by the aneurysm. The right axis deviation is readily understood, and as pointed out by Hirschboeck (1942), may have some diagnostic value when coupled with a water-hammer pulse. If the existence of such a lesion is borne in mind diagnosis after rupture is sometimes feasible.

Our thanks are due to Dr. Phillips, Medical Superintendent, Southmead Hospital, Bristol, for permission to publish this case: and to Professors Perry and Hewer for helpful criticism. We should also like to thank Mr. S. A. Edwards for the photographic reproductions.

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HEALED DISSECTING ANEURYSM

BY

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From St. Thomas's Hospital

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Since Shennan (1934) published his well-known report on 300 cases of dissecting aneurysm the literature of this condition has become extensive, and Sailer (1942) stated that there were then some 500 published cases, of which 33 had been diagnosed before death. There is general agreement that in some 80 per cent of these cases death occurs within a few hours of an acute onset, and that most of those patients who survive the accident of dissection are cardiac cripples who usually die of internal hæmorrhage or of cardiac failure within a year or two. There are, however, exceptions, such as Hall's (1926) patient, a boy aged 17, in whom the dissection occurred after a race, with such good recovery that he led a strenuous athletic life for 15 years. The survival record seems to be held by Graham's (1886) patient, who lived for 30 years after the dissection.

There is general agreement, too, as to the diagnostic criteria of the acute phase of the dissection. These have been well summarized by East (1939), who at the same time reports a personal case, diagnosed before death, of a woman, aged 43, who survived 5 years after the onset, despite a pressure of 300/150 during this period. That the healed dissection may be diagnostically obscure is pointed out by Gouley and Anderson (1940), who describe six patients in whom the onset was insidious and the clinical picture was that of cardiovascular syphilis. Pain was absent in four, inconspicuous in one, and occurred only intermittently for three weeks before death in one. All exhibited cardiac decompensation for periods varying between two months and four years. They all had big hearts, aortic regurgitation, and a dilated aorta radiographically: the Wassermann reaction was positive in one case only.

Others who have described aortic regurgitation with normal cusps in dissecting aneurysms are Resnick and Keefer (1925), Borger (1906), and Letulle (1905).

The case we now publish was, like those of Gouley and Anderson, diagnosed as syphilitic aortic reflux and aneurysm, though the possibility of a dissecting aneurysm was considered. The case is also of interest as illustrating the remarkable capacity for effort on the part of this victim of a healed dissection.

A Dutch engineer, aged 42, was first seen on March 19, 1942. He had gonorrhœa in 1921; his Wassermann reaction was then negative. In 1936, when aged 36, he played a long set of singles at lawn tennis in Holland and became so abnormally distressed that he had to stop. A week later, at 9 p.m., he was seized with very severe epigastric pain, which radiated to the angles of the scapulæ, more severely on the right side. He said that for two or three nights he was walking about in agony, and that morphia had to be injected, and that the pulse was said to have been very irregular: renal colic was diagnosed. He stayed at home for a fortnight, and subsequently resumed work, after which he noticed a tendency to dyspnœa and palpitation on vigorous exercise. In February 1937 he was investigated in a Dutch hospital; the aorta was "enlarged" and though the "blood test" was negative, he was given a course of intravenous arsenic. After this he was well enough to swim, and to play tennis, but not with his former vigour.

In 1940 he escaped from Holland on foot with his wife and children, carrying much baggage. He was able to swim and play tennis after his arrival in England that summer.

During January and February 1942 he became increasingly dyspnœic on exertion, and there was paroxysmal dyspnœa at rest. When examined on March 19, 1942, he was a

well-built man, not dyspnœic at rest, and presenting no evidence of congestive failure. Pulsation was seen and felt to the right of the sternum, where there was a diastolic shock. The presence of dilated venules along the line of diaphragmatic attachment suggested early vena azygos obstruction. The pulse was 92, with frequent premature beats; the blood pressure 140/60; and the arteries not unduly thick. On auscultation, a loud diastolic murmur was heard at the base and down the left sternal edge; the murmur was louder to the right than to the left of the sternum. No tracheal tug was felt. The knee jerks were brisk, and the pupils a little sluggish to light and not quite circular. On X-ray screening, the whole thoracic aorta was diffusely dilated. There was a considerable degree of left ventricular enlargement (Fig. 1), and the trachea was displaced to the right. His electrocardiogram showed numerous right ventricular premature beats, but was otherwise normal (Fig. 2, A).

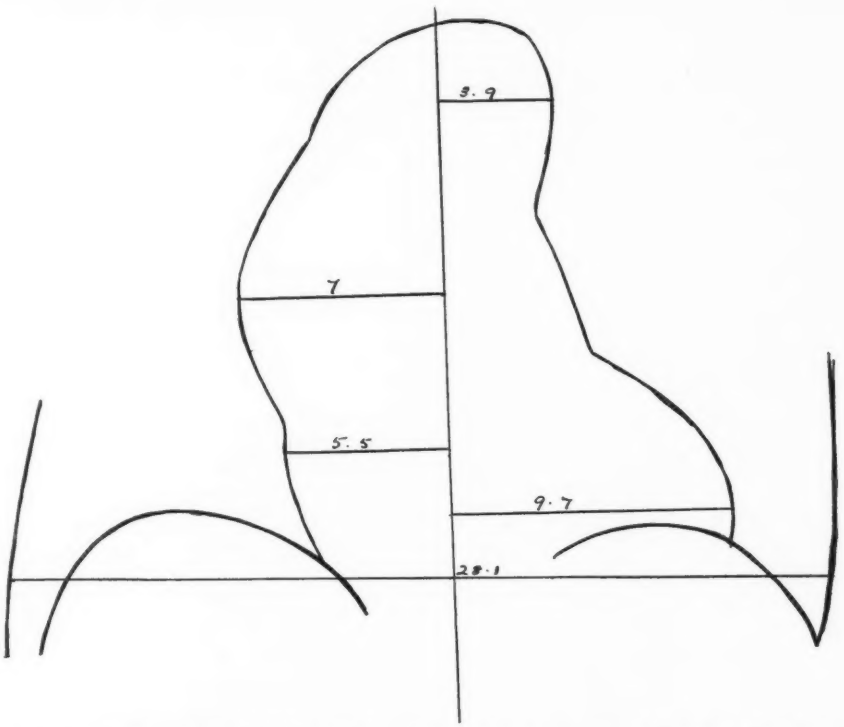
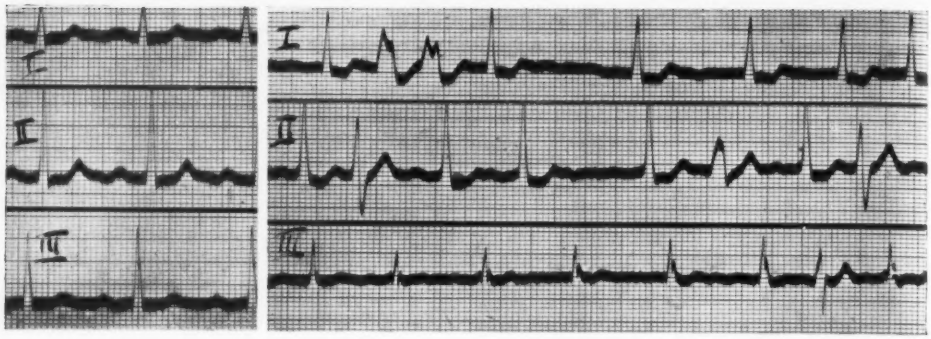


FIG. 1.—Orthodiagram on 25/5/42, showing left ventricular enlargement and dilatation of the aorta.

The diagnosis was aortic aneurysm, with aortic regurgitation. In view of the acute onset after violent physical effort, the possibility of either a ruptured valve or of a dissecting aneurysm was discussed. The latter diagnosis was considered to be improbable, because, apart from the onset, the clinical picture was typically that of a non-dissecting thoracic aneurysm. Moreover the radial and femoral pulses were equal, and it was thought that the combination of a dissecting aneurysm with free aortic regurgitation would hardly allow such a degree of physical activity as the patient had proved himself to be capable of. In spite of the fact that his Wassermann reaction was negative he was treated with mercury and iodide, and subsequently with stovarsol orally. When seen on May 25, 1942, he was working at his office full time, dyspnœic only on hills, and sleeping well with only one pillow. Dr. Anwyl Davies thought that in spite of the negative W.R. the condition was almost certainly syphilitic and advised further treatment with mercury and iodide.

In January 1944 he was still hard at work and almost symptomless, except for what



A B

FIG. 2.—Electrocardiograms. (A) Normal rhythm on 24/1/44. (B) Auricular fibrillation with ectopic beats on 21/5/44.

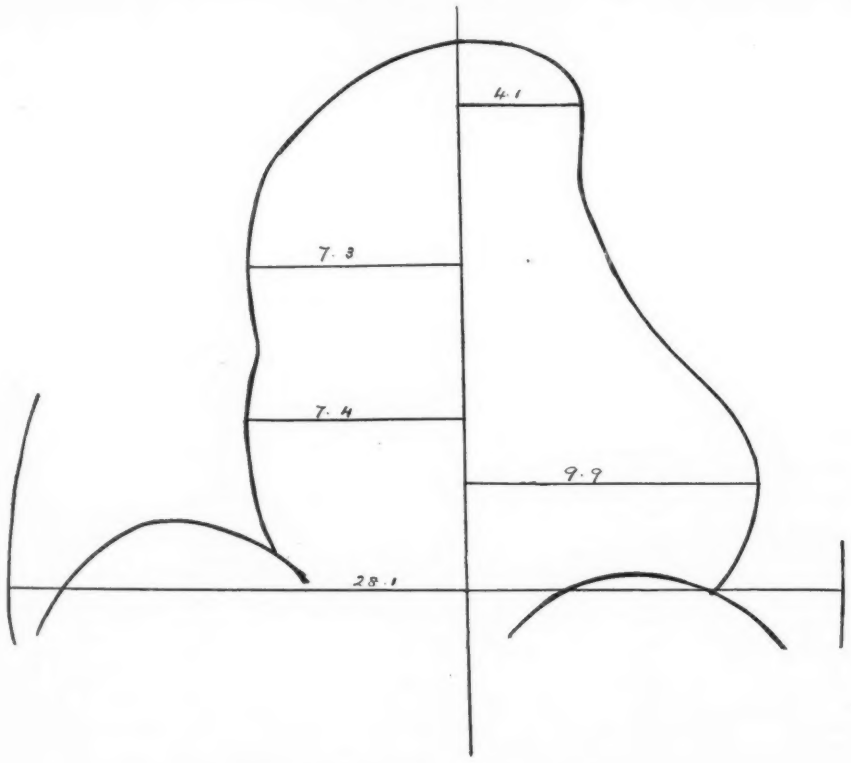


FIG. 3.—Orthodiagram taken on 9/1/45, showing further enlargement.

sounded like a paroxysm of auricular fibrillation during influenza a month previously. He had spent some months in Sweden, flying there and back under war-time conditions. Radiographically both heart and aorta were a little bigger. The electrocardiogram was unchanged.

In April 1944 there were further paroxysms of auricular fibrillation, lasting about two hours, and his condition had deteriorated when seen on May 21, 1944. Fibrillation had now become permanent, with multiple ventricular ectopic beats (Fig. 2, B). He was strongly advised not to follow the army of occupation into Holland, but felt that his knowledge of

the Dutch canals and their pumping stations was so unique that he insisted on going. He went by air to Brussels in September 1944. On his return he was able to work in his office only two or three days a week. He was increasingly dyspnœic on slight exertion, but his ventricular rate was easily controlled by a small dose of digitalis, and there was no congestive failure. In January 1945 an orthodiagram showed some further enlargement to the right (Fig. 3). Subsequently his condition deteriorated rapidly, and on March 4, 1945, he was admitted to St. Thomas's Hospital on account of increasing paroxysmal dyspnœa without evidence of congestive failure. On March 17, at 9.30 p.m., there was a sudden onset of sternal pain with severe dyspnœa, rapid pulse, sweating, and pulmonary œdema. He improved under morphia, but died within a few minutes at 10.30 a.m. on March 18.

Autopsy Report

The body was that of a well-nourished adult male.

Heart. A moderate excess of straw-coloured fluid was present in the pericardial sac. The heart was greatly enlarged, both sides showing the increase, but particularly the left. On section, the left ventricle showed hypertrophy, the thickness of its wall varying between 1·5 and 2·0 cm. Its cavity was dilated, the maximal internal diameters being approximately 7 cm. horizontally and 9 cm. in the superior-inferior direction. The right ventricle showed these changes to a corresponding degree, the thickness of its wall varying between 0·6 and 1·0 cm., and its internal diameters being both approximately 8 cm. The ventricular muscle appeared homogeneous and of normal consistency. The left auricle contained a thrombus in its appendix of approximately 1·5 cm. in diameter.

The tricuspid and pulmonary valves were normal. There was slight thickening of the edge of the posterior cusp of the aortic valve, and atheroma of the aortic cusp of the mitral valve. The aortic ring appeared dilated, its diameter being about 3 cm. The site of the intra-pericardial part of the aorta was occupied by a soft spherical mass having maximum diameter of 7·5 cm. (Fig. 4). The swelling lay superior to the right ventricle and anterior to

Fig. 4.—Heart and intra-pericardial aneurysm (anterior aspect). (A) Aneurysm.

the right auricle, compressing the latter posteriorly. Its surface was smooth, shiny, and for the most part of pale, blue-white colour. The mass was not adherent to any of the surrounding cardiac tissue except at the line of junction.

The coronary arteries were normal except for very slight patchy atheroma.

Aorta. On section, a horizontal rent in the intima was seen, extending from above the middle of the left posterior cusp round the left side across the anterior surface to the right lateral aspect, terminating just posterior to the union of the anterior cusp with the right posterior one. The maximal width of the rent was 2·5 cm.; its length was approximately 8 cm. The edge of the rent above the left posterior cusp was formed by free intima, which extended for about 3 cm. along its length. Here the floor was recently formed blood clot, part of the surface of that filling the intra-pericardial aneurysmal dilatation (Fig. 5). For the rest of the rent, the intima was bound by adhesions in an irregular manner to the media,

FIG. 5.—The same after opening. (A) Left ventricle. (B) Aorta. (C) Aperture into aneurysmal cavity showing surface of blood clot within.

FIG. 6.—More detailed picture of the split. (A) Left ventricle. (B) Rent in aorta, showing adhesion of split intima to underlying media.

which in this area also formed the floor (Fig. 6). The maximal width of the intimal tear was at the site of the visible portion of the blood clot and the adjacent area of the medial floor.

At the line of contact of the above-mentioned blood clot and medial floor, i.e. approximately superior to the junction of the anterior and left posterior cusps, there was free space which communicated superiorly and slightly to the right with a dissection in the posterior part of the ascending aorta. That part of the vessel immediately inferior to this free space showed slight saccular dilatation and gross irregularity of surface, much of which was due to localized atheroma.

It was apparent that the free space above mentioned formed the path of flow for the blood into the dissected false passage, and the small saccular dilatation and roughening of the aortic surface below was brought about by the impinging of the blood here on its passage through. The aneurysmal dilatation into the pericardium and the rent with a free intimal edge indicated a much more recent change.

The dissection, which initially lay posterior, tracked slightly to the left as it progressed upwards. The aneurysm thus formed posterior, inferior and right lateral relationships. The left lateral relationship was formed by the left pulmonary artery. The diameter of the dissection was here approximately 3 cm.

In the region of the arch of the aorta the false passage lay posterior and slightly inferior. There was only very small penetration upwards round the beginning of the innominate, left common carotid and subclavian arteries, reaching a maximum of 0·5 cm. These vessels thus formed the most superior border of the false passage (Fig. 7). As the aorta curved down-

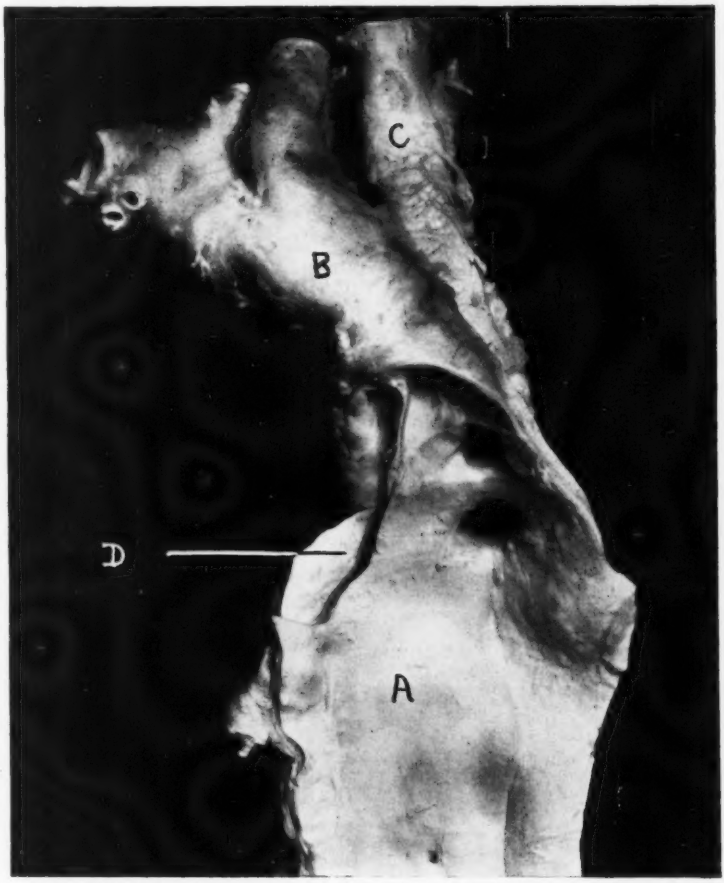


FIG. 7.—A view of the aorta further on. (A) Descending thoracic aorta. (B) Innominate artery. (C) Left common carotid artery. (D) False passage.

wards the false passage changed its posterior and inferior relationships to a posterior and slightly right lateral one, but when the diaphragm was reached it lay exactly posterior. At the bifurcation of the abdominal aorta the false passage lay right lateral (Fig. 8). Here the true aorta appeared continuous with the left common iliac artery, whereas the false passage descended down the right one for approximately 4·5 cm., 0·7 cm. above the division into the internal and external iliac branches. The site where the false joined the true arterial channel was clear cut. The former lay at first right lateral and then anterior to the first part of the true right common iliac channel, with the result that the free edge of union lay posterior to the former and anterior to the latter. The opening of the true right common iliac artery into the aorta had become displaced as a result of the dissection, appearing as a slit-like branch of the aorta where it became continuous with the left common iliac artery.

The false passage showed atheroma with ulceration and calcification on the wall not shared with the true passage, the change being most severe in the right common iliac artery 1·5 cm. up from the site of junction with the true iliac artery. It extended, however, also upwards to a point approximately 4 cm. from the bifurcation. There was no

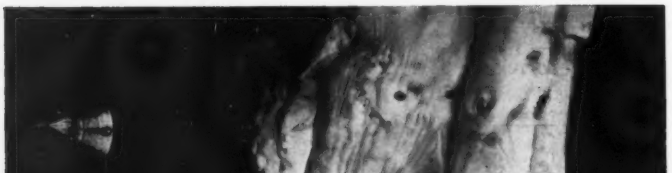


FIG. 8.—Bifurcation of the abdominal aorta. (A) The distal end of the aorta. (B) Left common iliac artery. (C) Atheromatous plaque in false passage. (D) Line of union of false passage with the true right common iliac artery.

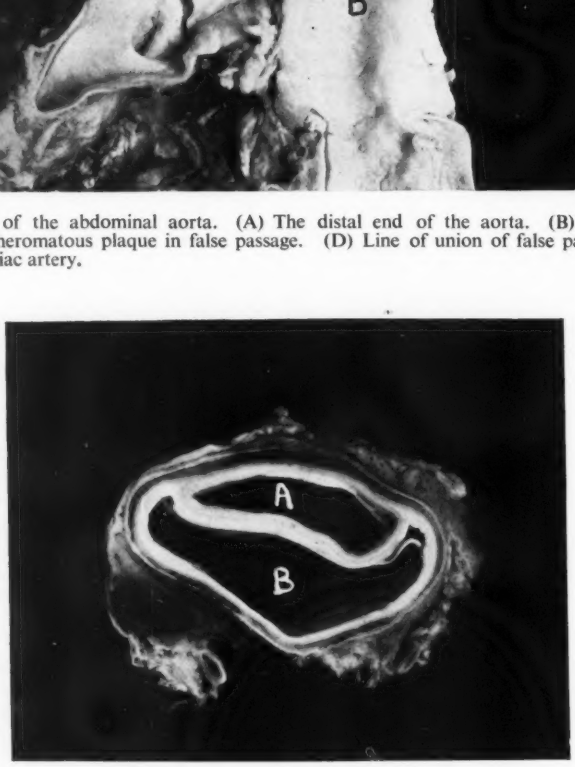


FIG. 9.—Cross-section of aorta at the level of the diaphragm. (A) True passage. (B) False passage.

change to a similar degree on the common wall in the corresponding places. The true aorta was also moderately atheromatous in all its extent. The posterior wall of the false passage showed in addition great unevenness of surface, raised white areas standing out from depressed more grey-white areas. There was also considerable wrinkling of the surface, particularly in a longitudinal direction.

Cross-section of the two channels at the level of the diaphragm revealed the internal diameter of the aorta when compressed to be 2 cm. and that of the false passage 3 cm. Thus there was a tendency for the latter to flank the former on its lateral aspects (Fig. 9).

The thickness of the true aorta varied from 0·15 cm. to 0·2 cm. The wall common with the false passage was only slightly less thick than the unsplit portion. The posterior wall of the false passage measured 0·10 cm. The cross-section of the common wall was clearly made up of three layers, a central yellow portion and slightly thicker light grey portions on either side. On the anterior aspect of the true passage the yellow layer became compressed and the inner light grey layer was proportionately more thick. The rest of the wall of the false passage was composed of light grey tissue only. The outer wall of the whole was covered by a very thin layer of softer pinkish-grey tissue.

The weight of the heart together with the intra-pericardial aneurysm and its contents was 1190 g.

Branches of aorta. The intercostal artery openings were patent in the true aorta, and some were linked with corresponding openings in the false passage by thin strands of fibrous tissue. The cœliac, superior mesenteric, inferior mesenteric, left renal, and left testicular arteries opened direct into the true channel. The right renal and right testicular joined the false channel, corresponding apertures being present also in the common wall. All the lumbar branches of both sides opened into the false passage. The corresponding apertures in the true passage were patent, and some of these, like the corresponding intercostal vessels, were linked by fibrous strands to the false passage openings. The middle sacral artery was not involved in the dissection and therefore opened into the true passage immediately above the bifurcation.

Respiratory system. There was a large excess of straw-coloured fluid in the left pleural cavity. Firm adhesions were present between the parietal and visceral pleuræ on the right side, especially in the basal region. A small excess of pleural fluid was present in this cavity also. The trachea and main bronchi were full of frothy straw-coloured fluid. The lungs (right—1021 g. ; left—794 g.) were extremely œdematous, but apart from patchy basal collapse, slight congestion, and emphysema they showed no further macroscopic abnormality.

The *liver* (2168 g.) showed moderate nutmeg change.

The *spleen* (298 g.) was firm, cut crisply, the cut surface being of deep purple colour.

The *left kidney* (383 g.) was congested.

The *right kidney* (326 g.), in addition to this change, contained an infarct in its upper half. The surface area involved was approximately 2 cm. square, having an irregular contour. The central part was light yellow and immediately surrounding it there was a zone of congestion. On section the infarct was shown to be wedge-shaped, the apex reaching the rim of the pelvis.

The remaining *abdominal organs* showed congestion.

There was a cortical adenoma in the *left adrenal*, 1·2 cm. in diameter.

The head was not opened. There was no macroscopic abnormality of mouth, larynx, pharynx, and thyroid.

Histology

Aorta. Sections were cut of the vessel (1) at the site of the rent and (2) just superior to the diaphragm. They were stained with hæmatoxylin and eosin, Van Gieson's stain, a modification of Weigert's elastic stain, and with Scharlach R for fat.

(1) The section was taken longitudinally through the aortic wall to include an area of the rent where the media formed the floor, and where the pulmonary artery was contiguous with the vessel. The media of the aorta here showed advanced degeneration. There was extensive fibrous replacement, diffuse mucoid degeneration and a few patches of necrosis. In one or two places the mucoid change had led to the formation of small lakes in the medial tissue. No fatty degeneration was demonstrated. The intima was a little atheromatous and the adventitial coat had undergone hyaline fibrosis.

(2) The split had taken place in the media approximately at the junction of the inner two-thirds and outer third (Fig. 10). The internal elastic lamina and the stripe of the true channel were not easily seen throughout the whole circumference of the vessel. The intima

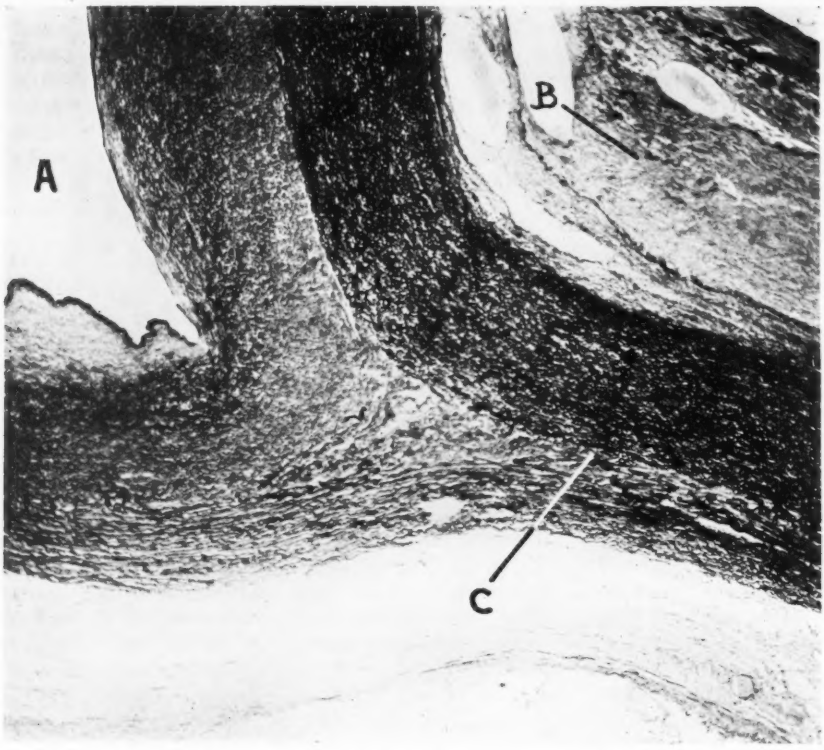


FIG. 10.—Photomicrograph of aorta at the level of the diaphragm (modified Wiegert's stain). (A) Lumen of false passage. (B) Intima of true passage. (C) The site of split in the media.

showed marked hyperplasia for about a third of its circumference, i.e. for the extent of nearly the whole of the wall not shared with the false channel. This hyperplasia was most marked between the internal elastic lamina and the stripe; the hyperplastic tissue showed also a hyaline degeneration. In the area of intima contiguous to the split in the media there were some fair-sized capillary blood vessels.

The false channel partly overlapped the true channel. The wall showed a variable thickness. The wall common with the true passage consisting of approximately two-thirds of the original media was thicker than that elsewhere. The thinnest portion was that diametrically opposite this common wall where there were only a very few elastic fibres. The media showed a progressive diminution in thickness between these two points. Under low power, in the section stained with hæmatoxylin and eosin, the false passage showed an inner layer of pale staining tissue, an intermediate zone staining light pink, the original media giving a deep pink, and lastly the outer adventitial zone. It appeared thus that a new " intima " had been formed. The elastic stain section showed that in both the inner and intermediate zones there was still considerable elastic tissue, the density of this becoming less as the lumen was approached, this diminishing content of elastic tissue accounting for the progressive lightening of the stain in the hæmatoxylin and eosin section. In both these zones the elastic tissue was very degenerate, contrasting sharply with that in the normal media. As a result of the high content of elastic in the new " intima " the inner surface of this false tube showed fine corrugation. Its surface was covered by a single layer of flattened endothelium. At that angle formed by the medial split which was not associated with intimal hyperplasia in the true passage there was a small valve-like projection from the " intimal " surface of the false passage. This consisted of a central core of dense elastic surrounded by a rim of less dense and degenerate elastic.

The adventitia surrounding the whole vessel was comparatively normal. A few elastic fibres were present. Collagen was a little more dense outside the free wall of the false passage where the elastic media was thinnest. The small vascular channels were dilated and full of blood and there had been hæmorrhage into the surrounding tissue. Round one of these vessels there was an accumulation of small round cells.

In summary, the histological picture confirmed the clinical conclusion that dissection down the aorta had occurred a considerable time before death. The period of active repair had passed and endothelium had formed on the surface of the false passage. Further, the appearance of atheromatous degeneration with surface ulceration and calcification in those parts of the false passage not shared with the true passage, without any corresponding change in the wall common with the true passage, is added evidence that the dissection was of old standing.

Kidneys. Both congested. Right kidney, four recently infarcted areas: an artery in relation to the largest of these contained a small recent thrombus. The capsule and the perirenal fatty connective tissue overlying the infarcted areas showed organizing chronic inflammatory granulation tissue. Adrenal congested with cortical adenoma.

Liver and spleen. Venous congestion, and slight fatty degeneration of liver.

Lungs. Slight congestion. Œdema. Emphysema.

Summary and Conclusions

A case of healed aneurysmal dissection of the aorta, of nine years' duration, is described. For some years the patient was able to live a very strenuous life, till symptoms of left ventricular failure appeared, the diagnosis then being syphilitic aneurysm with aortic regurgitation.

The autopsy findings are reported in detail.

There can be little doubt that the original dissection occurred in 1936, nine years before death. The diagnosis at this time was renal colic, and it is interesting to find that the right renal artery arose from the false channel. Renal colic and hæmaturia have been described in reports of acute dissections, which sometimes involve one or both renal arteries.

The histological appearances of the aorta at the site of the intimal tear conform with those of idiopathic cystic medio-necrosis of the aorta (Erdheim, 1929; Moritz, 1932; Roberts, 1939; and Davies, 1941). It is not unreasonable to assume that these changes were present to a degree at the time of the original accident.

Our thanks are due to Mr. A. E. Clark, for his assistance with the photographs, and to Dr. R. W. L. Todd, who referred the patient to us.

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Editorial Note on Another Healed Case

With the approval of the authors the notes of another healed case are added. Dr. A. G. Gibson has given me details of a woman where recovery was complete but unluckily she had no relatives from whom a history of the original illness could be obtained. She died from a cerebellar hæmorrhage and all that was known was that she had worked as a domestic servant for the last six years of her life without any serious illnesses or disability.

The whole aorta consisted of a double tube from just below the origin of the left subclavian artery to the left iliac artery. The figure (Fig. 11) shows the opening of the true aorta.

Autopsy by Dr. A. G. Gibson

A well-nourished woman, aged 46.

Heart. Hypertrophied. Sclerotic thickening of one aortic valve cusp. Atheroma at base of aorta and anterior leaflet of mitral. Both ventricles hypertrophied. No pulmonary embolism. Coronaries thickened but patent.

Thyroid. Large degenerating cyst in upper pole of l. lobe.

Aorta. Beginning from the descending part of the arch just beyond the left subclavian artery was another tube attached to the inner and posterior wall, heading down through the abdominal aorta to end in the left iliac artery one inch from its origin from the aorta. There was a wide opening at its origin in the aorta.

Lungs. Both collapsed.

Abdominal viscera. Normal except for some congestion.

Kidneys. Left: small, congested, slightly granular; arteriosclerotic scars. Right: diminished cortex; as left. Suprarenals. Both hypertrophied cortex.

Brain. Bleeding into both lateral ventricles (liquid). Clotted blood in the 3rd ventricle, in the iter and the 4th ventricle. Massive hæmorrhage in the middle part of the cerebellum which had reached the surface of the r. lobe. Some bleeding into anterior r. middle and posterior fossæ. Right sphenoidal sinus double. Lower one thickened mucosa and slight pus. Maxillary antra nil.

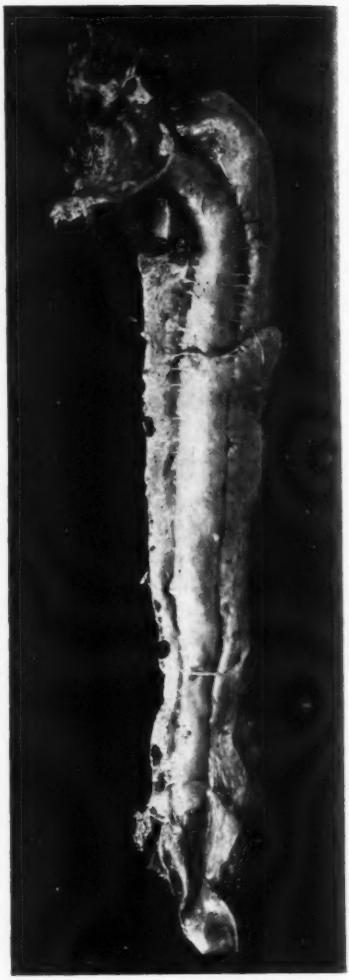


Fig. 11.—Photograph of the aorta, showing the opening of the true aorta and a healed dissecting aneurysm.

Maurice Campbell

SILENT DISSECTION OF THE AORTA

BY

JENNER HOSKIN AND FRANCES GARDNER

From the Cardiological Department of the Royal Free Hospital

Received March 27, 1946

Dissecting aneurysm of the aorta has been known as a pathological entity for over a century (Maunoir, 1802); it was first diagnosed during life by Swaine (1856), and a full account of its clinical and pathological features was given by Peacock in 1863. Even so, until recently only thirteen accurate ante-mortem diagnoses had been reported (Glendy *et al.*, 1937). Failure to diagnose the condition has been mainly due to its relative infrequency and to consequent lack of clinical suspicion. In a large series, Shennan (1934) reported one dissecting aneurysm in every 175 autopsies; Reich (1944) reported the average incidence as one in 380 autopsies, while in our own hospital it has been found only twice in the 900 autopsies performed during the last six years. In the last decade, however, clinicians have become increasingly aware of the condition and a further 84 cases, accurately diagnosed during life, have been reported.

Although about 80 per cent of patients die within the first few days of dissection, accurate diagnosis is important because, in a small number, life and moderate health may be sustained for some years with appropriate supervision. The classical picture of dissecting aneurysm has been repeatedly described (Peacock, 1863; Gager, 1926; and White *et al.*, 1935). The sudden onset of severe and prostrating pain, widely distributed throughout the body, together with evidence of interference with the blood flow in vessels arising from the diseased aorta, the appearance of an aortic diastolic murmur and a history of long-standing hypertension, form a syndrome which is not difficult to recognize once its significance is appreciated. Rare cases had, however, been reported where pain was absent and yet a recent or healed dissection was found at autopsy. These silent dissections are generally limited in extent, and it is not surprising that, in the absence of the characteristic pain, they are rarely suspected during life.

We report the following case because we have evidence of a silent dissection of the aorta. It was possible to make the diagnosis during life because the patient suffered a second attack characterized by the classical features of dissecting aneurysm. We believe this case to be of interest because the patient survived a second dissection of his aorta for over eighteen months, and because his illness was complicated by paroxysmal auricular flutter.

Case Report

The patient, a cinema agent of 56, was first seen by us in October 1942. He had been breathless on exertion for two years, but recently had grown so much worse that a walk of only a few yards would cause him severe distress. During these two years he had had attacks of palpitation ; these might come on at any time and usually lasted a few hours. On questioning, he admitted to occasional substernal discomfort but at no time had he had any severe præcordial pain. Four years earlier he had been treated by rest in bed for obesity and hypertension.

Examination showed an obese Hebrew with cyanosis; he was so breathless that the slightest exertion caused him real distress. The pulse rate was 140 a minute, the rhythm irregular, and the blood pressure 180/130 in both arms. The heart was enlarged; there was a triple rhythm and a harsh systolic murmur at the apex and a ringing second sound in the aortic area. There was some congestion at the right lung base.

X-ray examination of the chest revealed an enlarged heart and an enormously dilated and

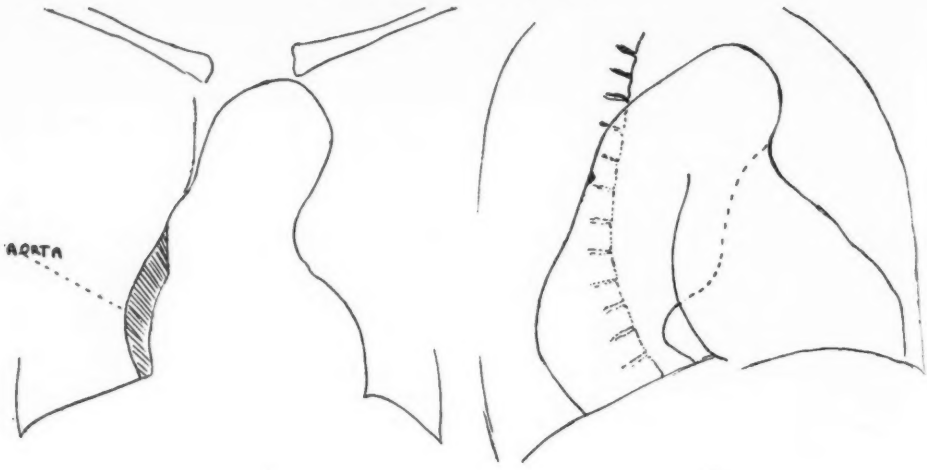


FIG. 1.—Orthodiagrams. 16/10/42.
(A) Showing dilated and tortuous aorta.
(B) Right (I) oblique position. The extent of the aortic enlargement can be clearly seen.

tortuous aorta (Fig. 1 and 2, A). The latter was seen more clearly on screening, when the lower thoracic aorta could be recognized lying behind and extending to the right of the right border of the heart (Fig. 1). The cardiogram showed irregular 2 : 1 and 3 : 1 auricular flutter (Fig. 3). The Wassermann and Kahn reactions were negative.

He was admitted to hospital and given digoxin, 0·25 mg. six-hourly by mouth. Normal rhythm was restored on the fourth day of treatment and there was sinus rhythm with depression of S–T II and III, compatible with digitalis therapy (Fig. 3, B). A diagnosis of atherosclerosis of the aorta associated with long-standing hypertension was made.

Thereafter he improved rapidly; the pulmonary congestion cleared and, when discharged three weeks later, he was no longer breathless when up and about. On discharge, quinidine sulphate 9 grains (0·6 g.) daily was prescribed, and with this treatment the improvement was maintained during the next few months. He was able to look after his own home but he did not return to work; the heart rhythm remained normal and the blood pressure was maintained at about 175/120.

In March 1943, while at some dog races, he was seized with an agonizing pain in the chest and left arm. He lost consciousness for about half an hour, and after recovery complained that the left arm felt numb and useless. He was taken to a hospital nearby, where, on admission, the left hand was found to be cold and limp though movements at the shoulder were preserved. The state of the radial pulse was not recorded. Two hours later a detailed examination was made; the left hand was now warm and perfectly normal. While in hospital the cardiovascular findings were as follows: pulse 74–130, sometimes irregular; B.P. 110/90; heart enlarged to the left with double aortic murmurs; no congestive failure; sinus rhythm with a rate of 74 a minute. He was discharged after eight weeks.

We saw him two months later. During this time he had been without quinidine and had noticed a return of the palpitation. On examination, the pulse was 140 a minute and regular, but by contrast with our earlier observations the blood pressure in the right arm was now 110/90 and in the left arm 110/100, while in the aortic area the heart sounds were completely replaced by to and fro murmurs. There was no pulmonary congestion or peripheral œdema. There was 2 : 1 auricular flutter; X-rays of the chest showed slight increase in the diameter of the lower thoracic aorta but no other significant change (Fig. 2, B and C). It was at this time that we realized that our original diagnosis had been incomplete; the man had a dissecting aneurysm and the recent episode was a second dissection. He was readmitted and again normal rhythm was restored after a few days' treatment with digitalis. Quinidine

A B C

FIG. 2.—Radiograms of the chest.

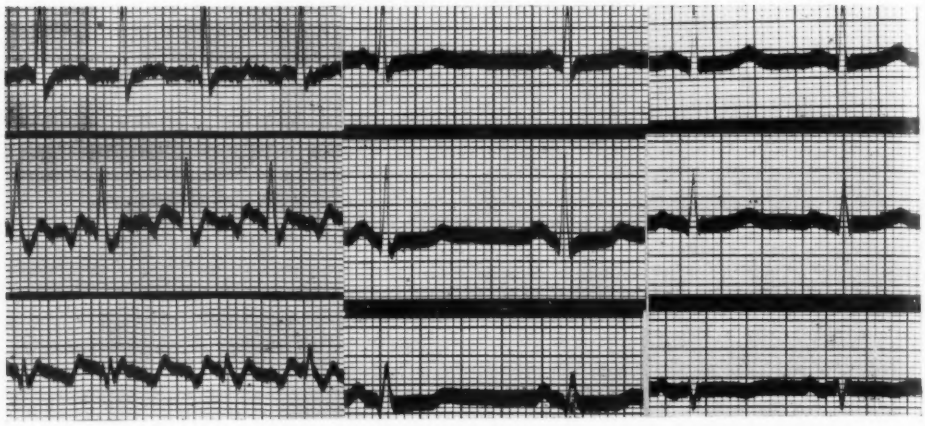
(A) 16/10/42. Postero-anterior view, showing the dilatation of the arch of the aorta and the lower thoracic aorta extending to the right of the right border of the heart.

(B) 15/7/43. Postero-anterior view after the second dissection, showing slight increase in the size of the lower thoracic aorta.

(C) 15/7/43. Right (I) oblique position after the second dissection.

therapy was reinstituted and subsequent cardiograms showed a sinus rhythm with low voltage T waves (Fig. 3, C).

He was discharged after two weeks, but from this time could only lead a semi-invalid life. He was often severely breathless and from time to time complained of a " boring " pain in the back of the chest. The physical signs and blood pressure remained essentially unchanged. In July 1944, early congestive cardiac failure developed and he was readmitted for a few weeks. He improved slightly, but after discharge the signs of failure steadily increased. In the evening



A B C

FIG. 3.—Electrocardiograms. Reduced to nine-tenths.
(A) 5/10/42. Showing 2 : 1 and 3 : 1 auricular flutter.
(B) 16/10/42. Sinus rhythm with slight depression of S–T I and S–T II compatible with digitalis therapy.
(C) 16/7/43. Sinus rhythm with low voltage T waves and slight inversion of T III.

of 20/12/44, after unusual exertion, he suddenly developed a very severe continuous pain in the back; it regressed to some extent, but a few hours later he died suddenly.

By the courtesy of the coroner for Westminster, we were able to attend the post-mortem examination. There was a partially healed dissecting aneurysm of the aorta, which had ruptured into the right pleural cavity.

Post-mortem findings

The heart was enlarged and the walls of the left ventricle were hypertrophied; the heart valves and the coronary arteries were healthy. In the aorta there was an elliptical opening in the intima 5 cm. above the aortic valves extending two-thirds of the way round the right and posterior aspects of the vessel. This orifice led backwards towards the heart into a large sac, filled with organized thrombus and lying in front of the superior vena cava and the right auricle. Viewed from the auricular aspect the sac was bulging into the cavity of the right auricle in front of the superior caval opening. From the elliptical tear the dissection wound spirally upwards in front of the ascending arch of the aorta, to reach the posterior aspect of the descending part of the arch, and in so doing the origins of the great vessels arising from the arch were dissected up 2–3 cm. The spiral direction of the dissection continued, the aneurysm lying on the posterior and the right aspects of the thoracic aorta, and it had ruptured back into the abdominal aorta, which was atheromatous, just above the bifurcation.

Throughout, the dissection occupied two-thirds of the circumference of the aorta, the true aorta appearing as a narrow tube attached to the larger dissecting aneurysm. Just above the diaphragm the aneurysm formed a saccular dilatation extending into the right pleural cavity. In this sac there was a longitudinal rent, 3 cm. in length, where the external rupture which caused his death had occurred. The walls of the aneurysm were covered with a pearly white

tissue resembling endothelium, with here and there partially organized adherent thrombi. The other organs showed no significant changes.

Discussion

The radiological appearance of the aorta remained essentially unchanged during the period this patient was under observation, and it is on this evidence that the diagnosis of silent dissecting aneurysm was made. The classical features of aortic dissection were only present during and after the second attack, and it seems likely that on this occasion the great vessels arising from the arch of the aorta became involved in the dissection, for the abnormal blood pressure readings found subsequently were evidence of new and permanent interference to the blood flow in the upper limbs.

The significance of the cardiac arrythmia found when this man was first seen is not entirely clear, but its presence supports the view that he had at that time a dissecting aneurysm. Abnormal heart rhythm has not often been reported in aortic dissection, probably because the common early fatal termination precludes cardiographic studies. In an extensive search we have found only one other report of auricular flutter associated with dissecting aneurysm (Mote and Carr, 1942), and other arrythmias are also rare. The saccular dilatation at the base of this patient's aorta encroached on the region of the sinus node and must have interfered with the mechanics of the right auricle. We therefore think the aneurysm was responsible for the auricular flutter, though, if this was so, it is remarkable, in view of the gross anatomical disturbance in this area, that digitalis and quinidine were able to control the abnormal rhythm.

Painless dissections of the aorta are rare episodes in a rare disease. In most of the reported cases the aneurysms have been limited in extent or they have failed to involve any of the great vessels arising from the arch of the aorta (Wedd and Thomas, 1932; Weiss, 1940; and Reich 1944). Sometimes the dissection has occurred during sleep and unconsciousness has supervened so rapidly that no complaint of pain could be made (Jessiman, 1939). In our case the original dissection was unusually extensive; it had probably involved the ascending aorta and from the X-ray appearances it is clear that the arch and the whole thoracic aorta were also affected. The absence of any history of anginal pain and the presence of hypertension in the arm suggest that this dissection had caused no interference with the blood flow in the coronary arteries or the great vessels arising from the aortic arch.

The pain of dissecting aneurysm has been ascribed to rapid distension of the adventitial tissues of the aorta by the effused blood and it has been thought that it may be absent if the dissection takes place slowly. In many cases, however, the pain, though severe, is limited to one arm or to both lower extremities, and in a few instances it has been indistinguishable from that of coronary thrombosis (Wainwright, 1944). Post-mortem examination in these cases has shown that the main limb vessels or the coronary arteries respectively have been involved in the aortic dissection. It is probable, therefore, that ischæmia is partially responsible for the pain and that in those rare cases where the coronary blood flow and the blood supply to the limbs is unimpaired, pain may be absent or inconsiderable.

For the majority of patients with dissecting aneurysm of the aorta no medical treatment can alter the fatal outcome. In a small number where, as in the case we have described, rupture back into the general circulation has taken place, complete rest in bed may relieve strain on the aneurysm wall and promote healing of the dissection. Subsequent management along the lines adopted for cardiac cripples does provide some hope of a return to partial activity.

Summary

A case of recurrent dissection of the aorta with auricular flutter is described.

The first dissection was silent and was survived for over two years; the second presented the characteristic syndrome, and the patient died eighteen months later.

The cause of pain in dissecting aneurysm is discussed.

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THE GENESIS OF THE NORMAL ELECTROCARDIOGRAM

BY

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The argument of this paper is based on the great work of Lewis, and all references to "Lewis, 1925" are to the third edition of *The Mechanism and Graphic Registration of the Heart Beat*. His demonstration (confirming Engelmann, 1875) that the contraction wave passes through cardiac muscle from the point of stimulation in all directions with equal velocity (p. 84); his considered view of the direction of the spread of the excitation wave in the human ventricles (diagram, p. 115); his table of synchronous events (p. 47); and his statement that the direction of travel of the contraction wave is of more importance than the disposition of contracting and resting masses of muscle in determining the form of the electrocardiogram (p. 57, footnote) are of fundamental importance.

In this paper two other points are emphasized: that air-filled lung is practically an insulator; and that the laws of composition and resolution of mechanical forces do not apply to electrical currents. On these concepts an explanation differing in some respects from that given by Lewis is founded.

The Fundamental Principle

If a battery, packed in insulating material, is placed inside a hollow shell of conducting material, with its positive and negative poles connected, through resistances p and n, with areas P and N on the inner surface of the shell, a current will flow from P to N through the wall of the shell in all directions (Fig. 1). If the terminals of a voltmeter are applied to any two points, A and B, on the surface of the shell, the voltmeter will register the difference of potential between them.

The heart is a battery: it is packed in insulating material, air-filled lung; its positive and negative plates are the anterior and posterior faces of an advancing contraction wave; and p and n represent the resistances of the conducting tissues leading from these "plates" to the wall of the thorax. The thorax is a hollow, hemi-ellipsoidal shell whose walls are made of conducting materials, viz. blood-filled muscles and, through the diaphragm, the liver. P and N are the most positive and negative points on the surface—the surface poles—and the string galvanometer is for all practical purposes a voltmeter, as it is standardized before each lead to register volts, not amperes.

Intrinsic and Surface Voltage

The difference of potential between two points on an electrical circuit is proportional to the resistance between those points. The total resistance of the circuit from the + plate to the − plate is $p+s+n$, where s is the resistance of the shell. If V is the potential difference between the plates—the intrinsic voltage of the battery—then the potential difference, E, between the surface poles P and N—the surface voltage—will be $V\left(\frac{s}{p+s+n}\right)$. If s is constant the relation of E to V depends on the values of p and n; if either is large, E will be small. But if these are constant and s variable, a decrease in s will diminish the ratio $\frac{E}{V}$

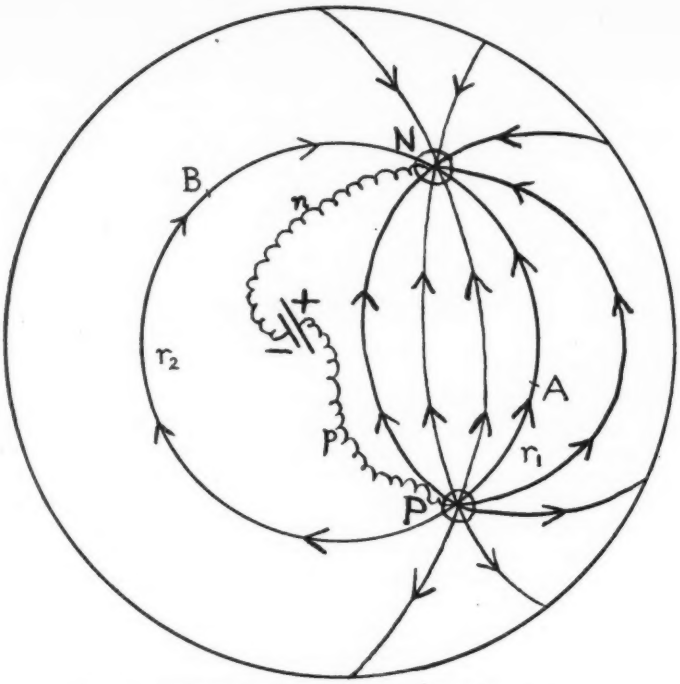

FIG. 1.—Diagram of a battery enclosed in a hollow shell.

+ −	Positive and negative plates of battery.
P, N.	Positive and negative " surface poles."
p, *n*.	Resistances between plates and poles.
A, B.	Points on the surface of the shell, connected by a voltmeter (not shown in diagram).
r_1.	Resistance between P and A on circuit P–A–N.
r_2.	Resistance between P and B on circuit P–B–N.

It can be shown mathematically that the resistance of a hollow metal sphere of uniform thickness and conductivity is practically constant wherever the poles are applied, unless they are very close together. If Fig. 1 represents a sphere of 36 in. circumference, and P and N are circular areas 2 in. in diameter, the resistance *s* will vary by less than ± 10 per cent from a mean value so long as the centres of P and N are not less than 4·5 in. apart. But at 3 in. the resistance is halved, and at 2 in. it is zero; for then the edges of P and N are in contact and the current is short-circuited without passing through the shell.

It will be seen that low surface voltage, without change of intrinsic voltage, may be due to poor contact between the heart and the chest-wall at one or both of the poles, to proximity of the surface poles, and to internal short-circuiting. These account for the low voltage of some phases of the normal cycle.

In pathological conditions low surface voltage (i.e. low voltage in all leads, including the most favourable chest lead) is sometimes thought to be evidence of degeneration of the myocardium or of interference with its function, e.g. by tamponnade. This is not necessarily the case. In advanced emphysema the interposition of a pad of lung between the heart and the chest wall may cause very low surface voltage for many years before there is any sign of heart failure. Pericardial effusion may have the same effect. It is possible that the low voltage associated with manifest heart failure may sometimes be due to the presence of fluid in the pericardium.

It will also be seen that the " surface zero "—the potential intermediate between P and N —bears no fixed relation to the " intrinsic zero." If $n > (s+p)$ the whole surface of the shell, including N itself, must be positive. No point or combination of points can be found on

the body that will give a true zero potential, or will be at the same potential throughout the cycle, or will be at the same potential at the same phase of the cycle in different patients. The various forms of two-point and three-point lead may prove clinically useful, but they do not give absolute voltage values, comparable from patient to patient, and it would surely be advisable to avoid terms that suggest that they do, such as unipolar lead, voltage lead, indifferent point, etc.

Potential distribution in the Normal Cycle

Fig. 2 to 6 and 8 show the main features of the potential distribution on the chest, with diagrams of the corresponding events in the heart. In constructing the diagrams of ventricular excitation the time-relations given by Lewis (1925) have been followed. The contraction wave is shown in the diagrams as a line between shaded and unshaded muscle. The shaded area is in contraction, and is conducting negative electricity from the posterior face of the wave; the unshaded part is about to pass into contraction, and is conducting positive electricity from the anterior face of the wave; arrows show the paths of conduction. In the surface charts the shaded areas are positive, the stippled areas negative; the poles are shown as clear spaces with a + or − sign.

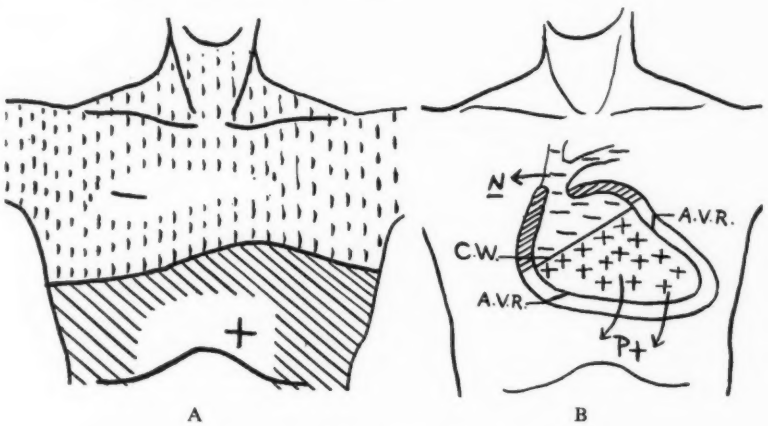

Fig. 2.—The P wave.
(A) Potential distribution on chest.
(B) Electrical events in the heart. A.V.R., auricular-ventricular ring. C.W., contraction wave. P, N, surface poles.

In Fig. 2B the wave has passed half-way down the auricles; these have begun to contract, and a wave of distension is starting to travel backwards from the superior vena cava. This distension wave reaches its maximum in the jugular veins during the inscription of QRS (Lewis, 1925)—the *a* wave of the polygram. It forms the negative pole. At the present stage its contact with the chest wall is poor—resistance *n* is large—and the surface voltage is low. There is also free internal short-circuiting through the intra-auricular blood.

In Fig. 3 the stimulus reaches both sides of the septum simultaneously. Both surfaces become negative, and there is no conduction path for the positive electricity in the interior to the chest wall except down the septum to the apex, where the positive pole is in immediate contact with a negative area that surrounds it. Shell resistance, *s*, is therefore minimal, and the surface voltage so low that there is no deflection.

The stimulus next reaches the papillary muscles (Fig. 4). There is not much short-circuiting, but again the poles are close to one another and the surface voltage is low.

The stimulus now strikes the endocardial surface of both apices and travels up both arborizations (Fig. 5 and 6). If the stimulus passes along the arborisation with velocity *a*, and at every point as it passes starts a contraction wave which spreads radially with velocity

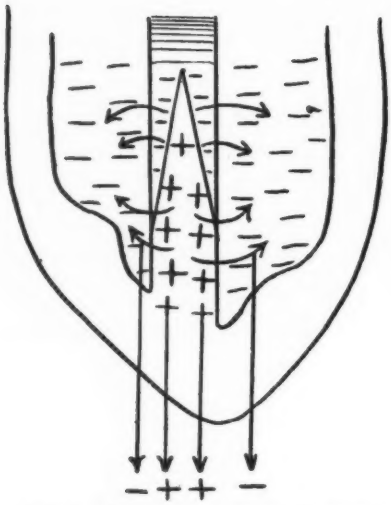

FIG. 3.—Stimulation of ventricular septum.

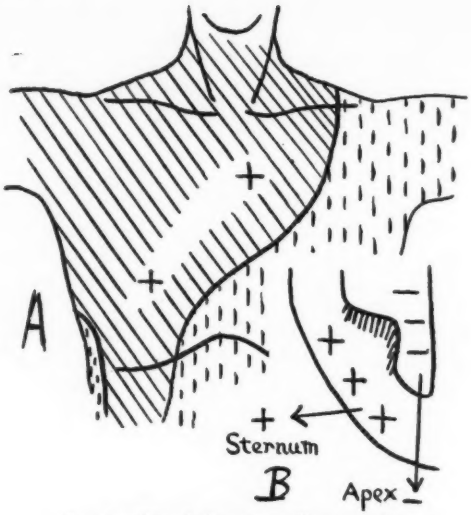

FIG. 4.—Stimulation of papillary muscles. (A) Potential distribution during inscription of Q. (B) (inset) Corresponding electrical events. The negative pole (not shown because of the inset) is at the apex.

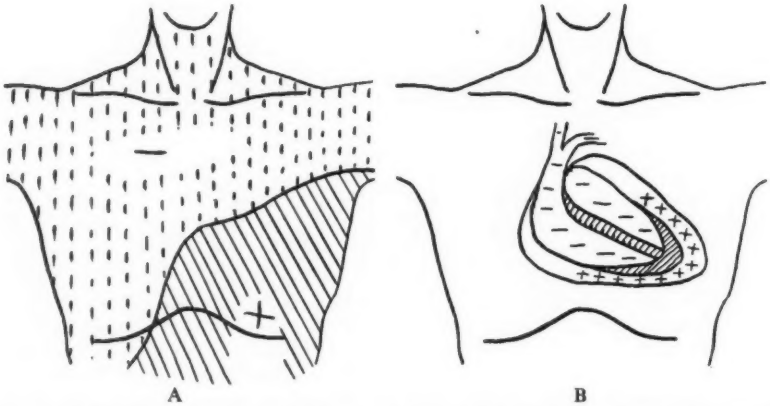

FIG. 5.—The first part of the RS deflection. (A) Potential distribution. (B) Electrical events.

c, the net result is a continuous contraction wave travelling away from the endocardium at an angle whose tangent is *a*/*c*. Meanwhile the distension wave has reached the innominate veins and is spreading across behind the manubrium from right to left. Both poles are now in close contact with the chest wall, and are not close together. *p* and *n* are small, *s* is large; the ratio E/V is at its largest and the surface voltage is high.

The left ventricle is thicker than the right, and the contraction wave takes a little longer to pass right through its wall. The effects of this are shown in Fig. 6.

On this view, septal contraction takes place during the last part of the P–R interval, the phase of silent activity. Comparison of normal and abnormal complexes in the same lead from the same patient shows that in the " short P–R, B.B.Bl. syndrome " (Hunter, Papp, and Parkinson, 1940) the part of the QRS deflection that precedes the notch does actually occupy this period. The features of this syndrome may be explained by supposing that there is a

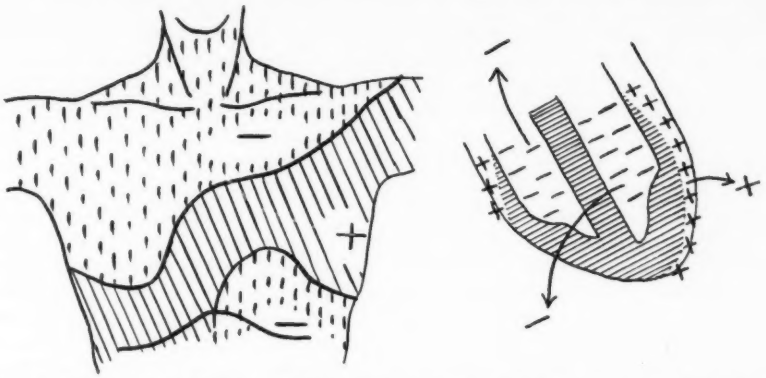

FIG. 6.—End of QRS deflection. Potential distribution and electrical events at the end of R I.

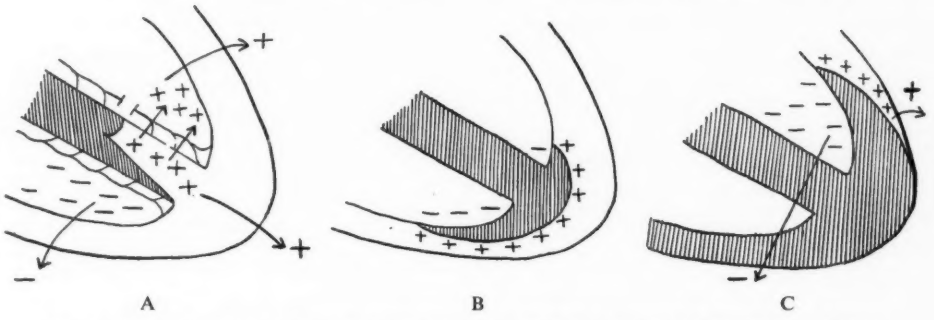

FIG. 7.—Sequence of electrical events during inscription of QRS in organic left bundle branch block. (A) The initial deflection. (B) The " notch." (C) End of the deflection.

shorter or longer delay in the passage of the stimulus down the left bundle branch. The first deflection is then due to unilateral stimulation of the septum; the form of the rest of the complex depends on the degree of asynchronism. Fig. 7 shows the sequence of electrical events in organic left bundle branch block; it is easy to see that these would give the typical electrocardiogram. This explanation has been suggested elsewhere (Hill, 1939) and independently by Roche (1945).

THE T WAVE

The only mystery of the T wave is its direction. When a strip of cold-blooded muscle is stimulated at one end the contraction wave deflection is followed after an interval by an anabolic wave deflection which is flatter, broader, and opposite in direction. The T wave follows the ventricular contraction wave after an interval, and is broader and flatter, but not necessarily in the opposite direction. There can be little doubt that it is anabolic; it is modified by many conditions that are known to affect anabolism—asphyxiation, ischæmia, diabetes, etc. Moreover, a ventricular extrasystole can occur immediately after it, or even during its downstroke, but never before it. This proves that some degree of anabolism occurs during its inscription.

But there is one form of T wave that does follow the pattern of the muscle strip—the T wave of a ventricular extrasystole; it is broader, flatter, and always in the opposite direction to the primary deflection. That it is an anabolic wave is proved by the fact that another ventricular extrasystole can follow immediately after it.

In the auricles mechanical contraction, i.e. diminution of cubic capacity with expulsion of contents, follows almost immediately on the electro-chemical event that causes the P wave; in the ventricles mechanical contraction begins during the S–T interval. Lewis' table of

synchronous events shows that the inscription of QRS is completed before the aortic valves open; QRS is synchronous with a sharp rise of intra-ventricular pressure to the level of the aortic pressure. During, and for some time after, this period the ventricular walls are put on the stretch, and the blood is squeezed out of them (systolic blanching can easily be seen in the exposed frog's heart). Shortly after the opening of the aortic valves the pressure in the aorta rises rather higher than in the ventricle; it is at this point that the T wave begins, and its peak coincides with the maximum difference of pressure at this stage. It comes to an end shortly before or after the aortic valves close ; the U wave accompanies the dicrotic rise in the aorta.

The distinctive feature of a ventricular extrasystole is that it causes little rise of intra-ventricular pressure; the walls do not become ischæmic, and the anabolic wave is able to follow its natural course in the wake of the contraction wave. With a normal contraction, anabolism is prevented by ischæmia; it has to wait for the return of blood, which penetrates inwards from the coronary vessels on the surface. It therefore travels in the opposite direction to the contraction wave, and, being of opposite sign, gives a similar deflection. It travels relatively slowly compared with the contraction wave, and most of the time the whole of both ventricles is taking part in it. Distension of the pulmonary artery and re-expansion of the right auricle give a wide distribution of negative potential, different from that obtaining during inscription of QRS, so that the T wave is not an exact copy of it in every lead (Fig. 8).

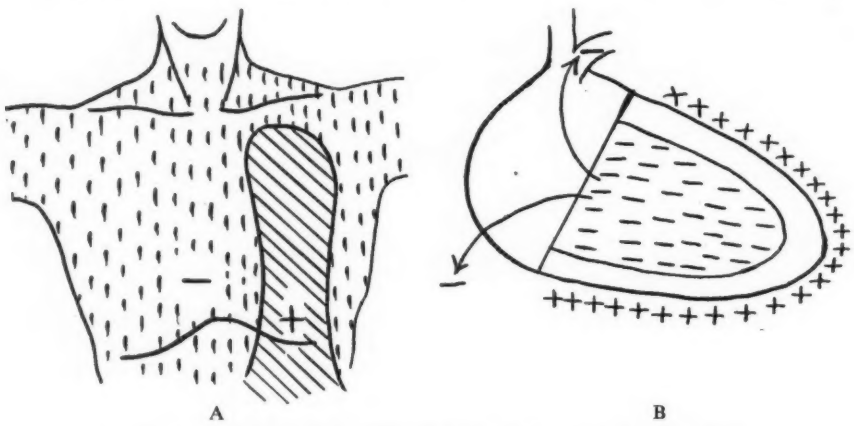

FIG. 8.—Peak of T wave. (A) Potential distribution. (B) Electrical events.

In short, the explanation offered is that the return wave of a ventricular extrasystole is the " natural " anabolic wave, and the normal T wave is the same thing modified by intra-ventricular pressure. When the aortic pressure during diastole is high and the left ventricle is beginning to fail its wall may be so much stretched that the coronary return is delayed till relaxation is far advanced; the T wave is then diphasic. Reduction of peripheral pressure by trinitrin reduces the intraventricular tension, and the T wave may then become upright and normal.

Delay of anabolism of the left auricle by intra-auricular tension may perhaps account for the onset of flutter or fibrillation in mitral stenosis.

INSCRIPTION OF DEFLECTIONS

In the normal subject there is a point at the costal margin, about three inches to the left of the middle line of the back, that is iso-electric with the standard left leg contact throughout the cycle. The left leg contact is virtually attached to the thorax at this point; similarly the forearm contacts are virtually attached to the thorax high up in the axillæ, the arms functioning simply as additional lengths of wire of low resistance.

It is convenient to think of the conduction paths between the surface poles, P and N, as

an infinite number of separate circuits lying side by side. Fall of potential between two points on a circuit is proportional to the resistance between those points. In Fig. 1, if E is the potential difference between P and N, R_1 the resistance of the circuit P–A–N, and r_1 the resistance of that part of it lying between P and A, the fall of potential from P to A is $E(r_1/R_1)$. If, by a similar notation, the fall of potential from P to B is $E(r_2/R_2)$, the potential difference between A and B, registered by the voltmeter, will be $E(r_2/R_2-r_1/R_1)$.

The positions of the surface poles during inscription of R in lead 1 are shown in Fig. 5A. The negative pole moves leftward to the position shown in Fig. 6, following the backward movement of the distension wave along the innominate vein. In Fig. 9 the left axilla corresponds with A in Fig. 1, the right axilla with B. Since the conducting musculature is much the same on both circuits it is clear that r_2/R_2 is at first much greater than r_1/R_1; conse-

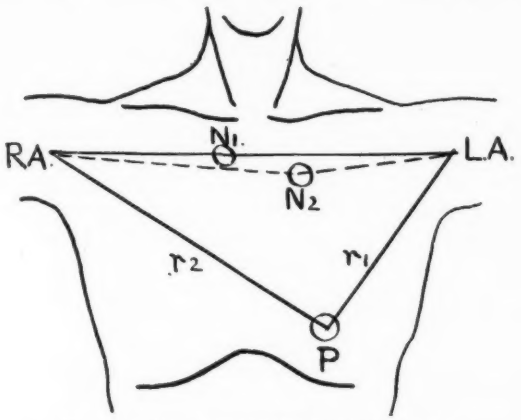

FIG. 9.—Diagram illustrating inscription of R I.

quently L.A. will be strongly positive to R.A., and there will be a large upward deflection. But as N moves leftward from N_1 to N_2, R_2 increases at the expense of R_1 while r_1 and r_2 remain unchanged. The difference between L.A. and R.A. therefore diminishes and the deflection falls back to the base line. A little further movement of N to the left would invert it.

Second and third lead deflections are determined by the same principles, but as the conduction paths from the left leg point pass through the dorsal muscles their amplitude and direction vary with the shape and muscular development of the chest.

ELECTRICAL AXIS

It is not too much to say that the whole doctrine of electrical axes, resultant and component currents, right and left preponderance, summation of currents, equilateral triangles, etc. is quite untenable and must be abandoned. The direction of an electrical current is determined solely by the conducting paths available and has no relation to the orientation of the battery in space. Electrical currents do not " sum " unless their sources are connected in series—the negative pole of one to the positive pole of the other and vice versa. When connected in parallel the voltage is intermediate between that of the two sources, the exact value depending on the arrangement of resistances, external and internal. Simultaneous occurrence of an auricular contraction and a ventricular event is equivalent to connection in series (combine Fig. 2B with Fig. 5B or 8B); the P wave appears superimposed unchanged on the QRS or T. But the two ventricles are connected in parallel, having a negative contact in common and a continuous positive pole (Fig. 5B and 8B); it is impossible that their voltages should be added.

It is true that if the ends of a diameter of a mass of conducting material are maintained at a potential difference V, a voltmeter applied to the ends of other diameters will show intermediate voltages resembling the components of a mechanical force; but the law determining

their magnitude would depend entirely on the shape of the conducting mass. A component of a mechanical force is proportional to the cosine of the angle between its direction and the line of the force, but this would be true of electrical components only if the mass were a straight rod of uniform cross-section. If the mass were a sphere the voltmeter would show a voltage equal to $kV \cdot \log\left(\frac{1+\cos A}{1-\cos A}\right)$; with other shapes other rules would hold good.

But when we recognize that air-filled lung is almost an insulator the picture becomes that shown in Fig. 1. Here the orientation of the battery matters not at all; all that matter are the points at which the paths of least resistance from the plates of the battery most nearly approach the inner surface of the conducting shell. These become the surface poles, and potential distribution then follows the ordinary laws of conduction through the walls of the shell.

It is, then, difficult to assign any precise meaning to the term electrical axis; but right and left axis deviation are convenient labels for two contrasted types of electrocardiogram.

Left axis deviation. The tall R I and deep S III indicate that at the peak of the QRS deflection the left axilla is strongly positive to the other two contacts; (similarly, the deep S I and tall R III of R.A.D. show that the left axilla is strongly the most negative of the three). Left axis deviation is common with enlargement of the ventricles, but has never been satisfactorily correlated with preponderance of the left ventricle (Lewis, 1928). All that is necessary for its appearance is that the positive pole, formed by the apical halves of the ventricles, shall be electrically closer than usual to the left axilla. The mere anatomical displacement of the apex is enough to secure this, but when the heart is much enlarged collapse of the base of the left lung must contribute to the effect.

The natural electrical action of the heart was imitated in a series of experiments by applying the poles of a battery (with suitable precautions) to various points on the chest of a subject attached to an electrocardiograph and noting the deflections resulting.

In the first experiment the negative pole was fixed to the middle of the sternum at the level of the second space. Applying the positive pole to the apex beat gave a large upward deflection in lead I and also in lead III; their relative amplitudes were comparable to those of R I and R III in the subject's standard cardiogram. Moving the positive pole to the sixth rib in the anterior axillary line inverted the deflection in lead III, giving the picture of L.A.D. Moving it inward, by centimetre steps, horizontally from the apex to the middle line gave smaller and smaller upward deflections in lead I, but no S I appeared till the middle line was crossed.

Fig. 10A, from a patient whose heart was seen by X-rays to be absolutely vertical and centrally placed, shows that this procedure is a fair imitation of the natural event. The first lead deflections are tiny, but not inverted.

A positive pole at the sixth rib in the anterior axillary line will give the picture of L.A.D.; a natural positive pole is in this position when the ventricles are enlarged; is any more elaborate explanation necessary?

Right axis deviation. When this is present the right ventricle is nearly always hypertrophied, but the converse is not always true. In the early stages of mitral stenosis and in advanced emphysema there may be right ventricular hypertrophy without a trace of R.A.D.

In the course of the experiment described above the conditions that would obtain in "right preponderance" were imitated in an exaggerated form. The positive pole was applied to points on the chest over the right ventricular contact, the voltage was at least ten times as great as the intrinsic voltage of any heart, and there was no positive pole at the apex to represent the left ventricle; yet no downward deflection could be produced in lead I as long as the negative pole was in the middle line.

In the second experiment conditions were reversed. The positive pole was fixed at the apex-beat and the negative pole moved to the left, centimetre by centimetre, at the level of the second space. The upward deflection in lead III increased rapidly, but in lead I it decreased, and at 4·5 cm. from the middle line it disappeared; 1 cm. further to the left gave a deep S I and a tall R III—the picture of R.A.D.

The same result was obtained with the positive pole 4 cm. to the left of the apex in the fifth space, the point of no deflection being 5 cm. from the middle line.

It is clear, then, that a negative pole a very little further to the left than the natural negative pole at the end of the normal R I will give a downward deflection in lead I; and there are two structures in this position, conductors of negative electricity at this stage, which, when dilated, come forward to approach the thoracic wall—the left auricular appendage and the pulmonary artery. The role of a dilated pulmonary artery was suggested by Dr. John Parkinson; it brings the R.A.D. of infundibular stenosis into line with this theory (O'Farrell, 1938).

When these structures are dilated the right ventricle is always hypertrophied, but again the converse is not always true. In early mitral stenosis the right ventricle may be enlarged before much dilatation of the left auricle and pulmonary artery is evident; in emphysema the appendage is not dilated, and the artery is covered by a pad of distended lung.

Fig. 10B affords an indirect confirmation of this view. The patient was a man of 31, with perfectly normal heart, arteries, and blood pressure. In the second left space was a pulsating tumour; the maximum pulsation was 7 cm. from the middle line, dulness to percussion extended 2 cm. further to the left and down into the third space. X-rays showed a solid tumour in contact with the base of the left ventricle and moving with it. The cardiogram shows extreme left axis deviation, but the peaks come rather later than usual, and coincide with the moment when the stimulus reaches the base of the left ventricle. At this instant positive electricity was conducted from the surface of this area through the tumour to the chest wall at the point of pulsation, making the left axilla strongly positive.

The last experiment described above reproduces the conditions present when the heart is displaced bodily to the left by deformity of the chest wall. Fig. 10C is from such a case.

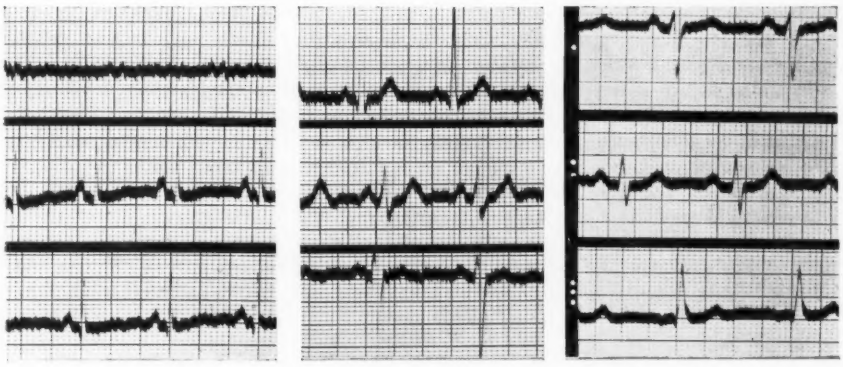

FIG. 10.—Electrocardiograms of: (A) Vertical, centrally placed, heart. (B) Left axis deviation due to an intra-thoracic tumour. (C) Right axis deviation due to displacement of the heart by deformity of the chest. (The second complexes in leads I and III of Fig. 10B have been traced over in Indian ink to facilitate reproduction.) (Reduced to two-thirds.)

X-rays showed no clock-wise rotation of the physical axis of the heart and no sign of hypertrophy of the right ventricle, nor was there any clinical reason to suspect this. The cardiogram shows marked R.A.D.

The argument may be summed up as follows.

1. While the negative pole is in the middle line no voltage applied to the left of the middle line can produce a downward deflection in lead I.

2. A negative pole in the second space 6 cm. to the left of the middle line reproduces the picture of R.A.D. even when the positive pole is to the left of the apex-beat.

3. There are structures, negative during QRS, which when dilated approach the chest wall at this point.

4. When these structures are dilated the right ventricle is always enlarged, but the converse is not always true.

5. When R.A.D. is present the right ventricle is nearly always enlarged, but the converse is not always true.

6. In both cases dissociation occurs in the same circumstances.

7. Exceptionally R.A.D. may be present without rotation of the physical axis of the heart or enlargement of the right ventricle.

8. A pathological positive pole 7 cm. to the left of the middle line in the second space produces extreme L.A.D.

Is it not reasonable to suppose that a pathological negative pole, known to be present in this region, may be the cause of R.A.D.?

Further, is there any direct evidence that the intrinsic voltage of a hypertrophied right ventricle is much greater than that of a normal left ventricle? or indeed of a normal right ventricle? If not, the doctrine of electrical preponderance has no basis whatever.

Salient points have already been discussed in the course of this paper; it only remains to emphasize that anatomical relations and physical principles are of paramount importance in the interpretation of electrocardiograms.

Summary

Fundamental principle. The heart is compared to a battery packed in insulating material and enclosed in a hollow conducting shell.

Intrinsic and surface voltage. The ratio of the maximum potential difference on the surface of the shell (the surface voltage) to the voltage of the battery (the intrinsic voltage) is determined by resistance relations. The low voltage sometimes found in emphysema and with pericardial effusion is thus explained.

Potential distribution in the normal cycle. Charts of this, with diagrams of the corresponding events in the heart, are given for each deflection.

The T wave. The T wave of a ventricular extrasystole corresponds to the secondary wave of a stimulated muscle-strip, and the normal T wave is the same thing modified by intraventricular pressure. Reasons are given for believing it to be anabolic.

Inscription of deflections. The principles determining the direction and amplitude of deflections are explained.

Electrical axis. The laws of composition and resolution of mechanical forces do not apply to electrical currents. Left axis deviation is ascribed to proximity of the positive apex to the left axilla; right axis deviation to an abnormal negative pole between the sternum and the left axilla, formed by dilatation (or occasionally displacement) of the left auricular appendage or pulmonary artery or both. These conditions can be imitated by applying the poles of a battery to appropriate areas on the chest.

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HEART-VECTOR AND LEADS

BY

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The purpose of electrocardiographic investigation is to find out something about the heart itself by means of an accurate knowledge of its electric action. This may be represented by the so-called heart-vector, a directed quantity, indicating in which direction electricity is propagated by the heart. The amount of each lead at a given instant depends on the heart-vector. The latter changes during the heart beat in direction and magnitude and, consequently, the value of the lead, too, changes with time.

The relation between heart disease and leads is, at the root, very complicated. One can, however, analyse this relation by dividing it into a relation between heart disease and heart-vector and a relation between heart-vector and leads. *The first relation is only to be found on the ground of medical experience. The second relation, on the contrary, is of a purely physical nature, and will be the subject of our discussion.*

A long time ago Einthoven *et al.* (1913) with his so-called triangle rule, tried to formulate the relation between the heart-vector (which he called the manifest potential difference) and the three limb leads. Einthoven himself realized very well that this rule gives only an approximation of the truth. What the significance is of this approximation can be decided only by measurement. We have tried to formulate the physical problem and next to obtain, by measurements on a phantom, the data necessary for the application of the fundamental solution of the physical problem.

The Relation between Heart-Vector and Leads

In the first place one must try to get a clear idea of the heart-vector. The propagation of electricity by an excited muscle has, without doubt, its origin in the electrolytes present in the tissue. By diffusion of electrolytes a potential difference is generated. This can be explained as a consequence of an electric field, acting on the ions. This electromotive field, together with the common electrostatic field, is the cause of an electric current in the electrolyte. In the case of living tissues, the cell-membranes have a preponderating influence on the generation of the electromotive field.

By the excitation of the heart muscle, an electromotive field arises. This is located in the heart muscle and causes a current in it as well as in the trunk around it. The heart muscle is the active part; the conducting tissue of the trunk the passive one. This can be compared to a galvanic cell (heart muscle) with an external resistance (rest of trunk). There is, however, a great difference here, because we have not to deal with a current through a wire, which can be characterized by one single number, the current intensity. In the trunk there is a *current-field.* We can represent this field by drawing current lines, indicating from point to point the direction of the current (Fig. 1). This figure holds only for one single instant, the current-field changing periodically with the heart beat.

Each little piece of the heart muscle contributes to the current-field independently of its other parts. This contribution is proportional to the volume of the small piece and to the intensity of the electromotive field existing in it. The total action of the heart muscle is the result of the actions of all the small pieces.

We must bear in mind that the action of each piece, i.e. its contribution to the total

current-field, depends also on its relative position in the heart. For simplicity, however, we shall for the time being ignore this complication. The current-field will then be determined by the (vectorial) sum of the electromotive field strengths in the different pieces of the heart muscle. This vectorial sum determines, by its direction and magnitude, the current field in the trunk. It is this sum that we have to call the heart-vector.

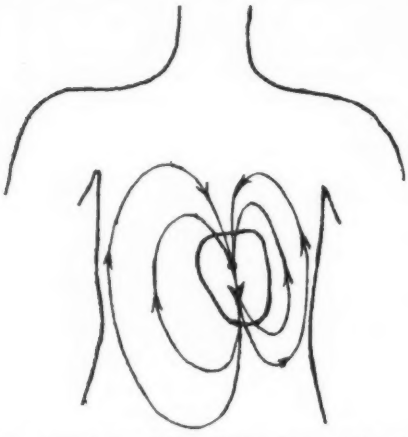

FIG. 1.—Current field in the trunk, caused by the electric action of the heart.

It is possible to give a generally valid relation between heart-vector and current field. The latter is the resultant of the fields that would be set up by the rectangular components X, Y, Z of the heart-vector separately. Each of these fields is proportional to the component by which it is originated. The current field in the trunk gives rise to potential differences between the points at the surface of the body (leads). The relation between these potential differences and the components of the heart-vector is of the same type as that between current field and heart-vector. Each lead, therefore, can be represented by the single equation :

$$\text{Lead} = aX + bY + cZ \quad . \quad . \quad (1)$$

a, *b* and *c* depend on the shape, dimensions, and conductivity of the trunk and on the position of the electrodes used. They do not depend, however, on the direction and magnitude of the heart-vector nor, therefore, on its components X, Y, and Z. During the heart beat the heart-vector changes and, therefore, its components X, Y, and Z change too; during this change, the constants *a*, *b*, and *c* remain unchanged.

We need three equations to solve the three unknowns X, Y, and Z, so that we have to measure three independent leads. These three equations contain 9 (3×3) constants of the type *a*, *b*, and *c*.

Between the three limb leads (LR, RF, and FL) there is a relation, their sum being zero, so that they are not sufficient to find the three components X, Y, and Z. In practical electrocardiography, however, the component that points forward is often neglected. The leads LR, RF, and FL would then, theoretically speaking, be sufficient to determine the two components of the heart-vector that are parallel to the frontal plane, but in reality the component that points forward may not be neglected.

The relation between heart-vector and leads may be represented geometrically, but instead of the equilateral triangle according to Einthoven, an oblique triangle has to be used. We shall not enter further into this geometrical representation here, nor shall we explain how the shape of the triangle depends on the coefficients of equation (1).

MEASUREMENTS ON A MODEL OF THE HUMAN BODY

Numerous investigators have tried to study the relation between heart-vector and leads with the aid of a model. They have employed models that were only very rough approximations of the human body. For example, triangular and circular pieces of filter paper, soaked in an electrolyte and glass tanks filled with an electrolyte have been used (Hess, 1935; Hollmann, 1937; Momm, 1933; and Wilson, 1930). It is very important that the flow of electricity through the body is a three-dimensional phenomenon, which cannot be studied by means of the electric current in a flat layer of liquid. The shape of the boundary of the conducting liquid influences essentially the course of the lines of flow. We have used a glass phantom, the shape of which is a copy, as correct as possible, of the human body on a scale of 1 : 3. At the ends of the extremities it is fitted with copper electrodes and it is filled with a diluted solution of copper sulphate. The cranial roof is absent ; through this opening the phantom is filled with the electrolyte and an artificial heart is introduced.

By choosing the correct shape of the phantom, only one requirement is fulfilled, namely, the one of the three-dimensional current. The other requirement is that the specific resistance from point to point, apart from a proportionality factor, be the same as in our body. The mean specific resistances of the different parts of the body do not diverge much, but especially for the trunk these differences are important (Hess, 1935). It is more particularly the lungs, with their high specific resistance, that account for an appreciable lack of homogeneity. In this first paper we shall deliberately neglect this complication and operate with a homogeneous trunk.

As an artificial heart we used two circular copper plates P_1 and P_2, both about 2 cm. in diameter (Fig. 2). These plates are parallel to each other and are connected with the ends of an isolating cylinder of a length of 2 cm. The isolated wires, let through a narrow glass tube B, serve to set up a potential difference between these plates. This potential difference gives rise to a current in the surrounding liquid. Fig. 2 represents a heart, causing a vertical heart-vector. Besides, we used also a heart of which the two plates are put vertically in the standing trunk and by which we can make a horizontal heart-vector act. We have only concerned ourselves with the leads LR, RF, and FL from the extremities. The component of the heart-vector pointing forward is neglected as usual.

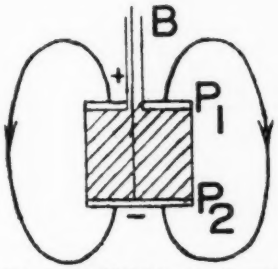

FIG. 2.—Artificial heart. P_1 and P_2=copper plates, being used as electrodes. B=tube, in which are connecting wires.

In the relevant papers we could not find reliable data concerning the position of the human heart. We used, therefore, rœntgenograms. The individual data diverge strongly; we used a mean position.

In our simplified case the relation between the two independent leads from the extremities and the two components of the heart-vector is given by two equations, containing four coefficients. In order to determine these coefficients, we have used successively a horizontal and a vertical heart-vector of equal magnitude. By measuring the two leads in both cases, we are able to calculate relative values for the four coefficients.

There exists, also according to Einthoven's triangle rule, a linear relation between the leads and the components of the heart-vector. This relation can be deduced from the well-known geometrical construction. The coefficients found in this way turn out to be rather different from those found with our model.

Absolute Measurements. So far, we have restricted our attention to the relative values of both the heart-vector and the leads. The question now is to introduce the absolute values. For that purpose we must express the heart-vector as well as the leads in centimetre-gramme-second units or in units connected with these (e.g. volts). The leads are expressed in volts. The heart-vector is the product of an electromotive field strength and a volume. The unit in which it has to be expressed is thus found from the units of field strength and volume. The conception of field strength is connected with that of potential difference in such a way that the field strength is the quotient of the potential difference between two points and their distance. We shall, therefore, express the field strength, as usual, in volts per cm. (volt/cm.). The heart-vector is then expressed in a unit found by multiplying the unit of field strength by the unit of volume, that is, in volt/cm. $\times$ cm.^{3} = volt . cm^2.

It is easy to measure the leads, for the model as well as for the human body, in an absolute unit, i.e. in volts. The absolute value of the heart-vector of the artificial heart is calculated from its dimensions and its potential difference. Taking into account the ratio between the dimensions of the model and the human body, the heart-vector of the latter can be computed.

The maximum value of the heart-vector is reached at the time of the R deflection. With 1 mV. for lead I and 1·5 mV. for lead II, we find a value of about 1 volt . cm.^{2} for the maximum value of the heart-vector. This result, however, does not appeal strongly to us as we are not familiar with the quantity " heart-vector " nor with the unit " volt . cm.^{2} " The mean value of the electromotive field strength in the heart muscle has a more direct meaning for us. This is found by dividing the magnitude of the heart-vector by the volume of the heart muscle.

Taking for the latter 250 cm.^{3} we get for the mean electromotive field 4×10^{-3} volt/cm.

Perhaps even this value has no direct meaning for the reader. But if we assume that at one and the same moment this field strength exists in the whole muscle, we find the potential difference between two " poles " of the heart by multiplying the mean field strength by the diameter of the heart (circa 9 cm.). In this way we find about $9\times4\times10^{-3}$ volt$=$36 mV.

Influence of the Point of Application of the Heart-vector. The leads caused by the heart-vector depend not only on its direction and magnitude but also on its point of application. We placed the artificial heart there, where, according to the rœntgen data, the " centre " of the heart is situated. The heart, however, is not very small with respect to the trunk. We may, therefore, expect that the contributions of the different parts of the heart muscle to the heart-vector may not simply be added vectorially, as supposed in the preceding. One and the same electromotive field strength, acting in different points of the heart muscle, will give different contributions to a lead. In order to investigate how large these differences are, we put the artificial heart in different places in our phantom. For this purpose we chose not only the centre of the heart, but also the heart apex, the heart base, and the back of the heart. These selected points are situated on the surface of the heart, the last mentioned point lying straight behind the heart centre. The place of these points is found from radiological and anatomical data.

The results for the different places diverge appreciably. They give, however, a too unflattering impression, as we have chosen points on the surface of the heart, that is to say at a maximum distance from the heart centre.

Possible Application to Vector-cardiography

We may conceive the heart-vector as an arrow, drawn from an arbitrary point as origin. During the heart beat this vector varies as to its direction and magnitude and, in doing so, its end describes a curve. This curve is called by Schellong (1936) the vector diagram. This diagram repeats itself almost identically for each heart beat and represents a characteristic property of the heart.

The computation of the heart-vector for various phases of the heart beat is a laborious task. For this reason a direct instrumental method has been developed for visualizing the curve, the vector-diagram, or for photographing it (Hollmann, 1937*b*).

Up to now the vector-diagram, as far as it is determined from limb leads, is found with the aid of Einthoven's triangle rule. As a matter of course, by using our equation (1) we would find a different diagram from the same data. The vector-diagram found depends, namely, on the coefficients in these equations. The true vector-diagram is found by using the correct coefficients. Although our coefficients are not yet the correct ones, our method gives the means to approximate to the truth. In order to apply the correct coefficients in practice, the recording instrument should be provided with a device by which these correct values, found from the experiments on the phantom, are taken into account. The question as to which leads we must chose becomes in this way theoretically meaningless. For to each set of leads belongs a set of nine coefficients. If these coefficients have been taken into account in the recording instrument, we must necessarily find the true vector-diagram of the patient examined, a curve that represents the electric action of his heart. This curve must be independent of the choice of the leads. The latter is restricted only by practical considerations. This idea, however, has not yet been carried out experimentally.

Summary

There exists a linear relation between heart-vector and leads. The coefficients of the equation giving this relation can be determined by measurement on a model of the human body. This is filled with an electrolyte, in which an artificial heart is placed. As the poor electric conductivity of the lungs is not taken into account, the results are open to improvement.

The absolute value of the heart-vector can also be determined (R deflection$=$1 volt . cm.^{2}).

The influence of the situation of the excited muscle elements is investigated.

From the leads the heart-vector or a vector-diagram can be derived, either by calculation or by direct registration. The vector-diagram can be found from other leads as well as from the limb leads and will then turn out to be independent of the choice of the leads.

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THE HEART IN STERNAL DEPRESSION

BY

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The place where the apex beat appears on the chest wall depends as much on the symmetry of the thorax as on the size of the heart. A change in the alignment of the spine, the posterior fulcrum of the thoracic cage, in the form of scoliosis, alters the position of the beat. Local deformity of the ribs which form the walls of the cage will do the same thing. Deformity of the sternum, the anterior fulcrum of the thorax, as a cause of displacement of the apex beat has received less attention. The effects of depression of the sternum (pectus excavatum) on the shape and position of the heart have been studied in sixteen adults examined during the past year.

Description of Cases

All sixteen patients had been referred for an explanation of certain signs connected with the heart, with the knowledge that deformity of the chest was present, but without appreciating that the two conditions might be related. In many of them suspicion of heart disease had led to restriction of their physical activities and to a change of design for their future livelihood.

The *symptoms* that had caused the patients to seek medical advice in the first place were

TABLE I

Summary of Findings in 16 Healthy Subjects with Depressed Sternum

Case No.	Age	Sternal depression	Antero-posterior chest measurement in inches		Radiological findings in anterior view			
			Patient with control in brackets		Translucency of shadow	Apparent enlargement	Outward shift of left border	Scoliosis
1	36	Funnel	4½	(8½)	Absent	Absent	Great	Effective
2	15	,,	5	(8)	,,	,,	,,	Absent
3	57	Cup	5½	(8½)	Obvious	Moderate	,,	,,
4	19	,,	5½	(8)	,,	,,	,,	,,
5	19	,,	5½	(7½)	,,	,,	Moderate	Incidental
6	29	,,	5½	(8½)	,,	,,	,,	Effective
7	17	,,	5½	(7½)	,,	Moderate to slight	,,	Absent
8	17	,,	5½	(8½)	,,	,,	,,	Incidental
9	23	,,	5½	(7)	,,	,,	,,	Effective
10	36	,,	5¾	(8½)	,,	,,	Considerable	,,
11	15	Saucer	6	(7)	Slight	Slight	Moderate	Incidental
12	19	,,	6	(7)	,,	,,	,,	,,
13	19	,,	6	(7½)	,,	,,	,,	Absent
14	19	,,	6	(7½)	,,	,,	,,	,,
15	19	,,	6¼	(7)	,,	,,	,,	,,
16	31	,,	6¼	(8)	,,	,,	Slight	Incidental

Note. In addition, the apex beat was displaced outwards in every case and a systolic murmur was present. In the anterior view the pulmonary artery was prominent on cardioscopy in the funnel and cup depression, but not in the saucer variety. In the left oblique view the heart shadow was normal or smaller in size, and in each case it extended on to the spine.

FIG. 1.—Diagramatic representation of the position of the heart in the three kinds of depression of the sternum.

diverse and never the outcome of cardiac embarrassment. In none was there heart failure such as has been attributed to gross deformity of the spine (Coombs, 1930). The symptoms were irrelevant to the condition and were of a general kind such as palpitation, giddiness, tiredness, and lancing pain in the left chest. Of greater importance were the restrictions imposed on the patients, even when symptom-free, because of a routine medical examination. Rheumatic heart disease had been diagnosed in five cases, which were advised to submit to regular medical supervision. Four cases had been relegated to the low health categories of Grade III or IV by Medical Recruiting Boards. Termination of pregnancy had been advised for one. Long periods in bed and absence from her calling as a teacher had been enforced on two occasions in another, aged 57 ; while another, a boy of 19, had been compelled by his medical advisers to discontinue his studies as a medical student; and later was placed in Grade IV

FIG. 2.—Funnel depression in Case 1. Antero-posterior (A–P) measurement of 4¼ inches against 8¼ inches in control.

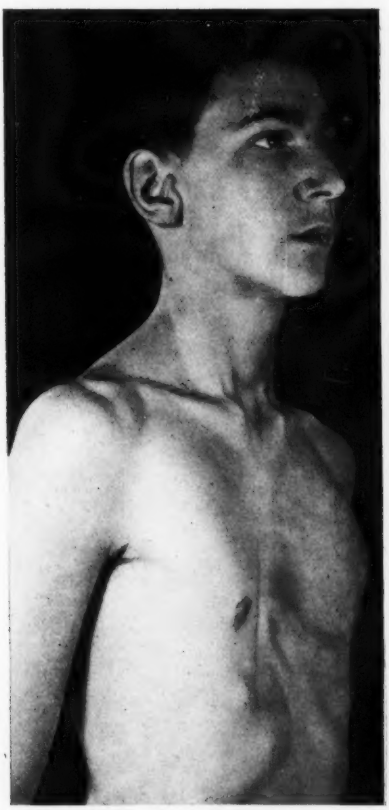

FIG. 3.—Funnel depression in Case 2. A–P measurement of 5 inches against 8 inches in control.

by three different Medical Boards after an examination that was amplified by a radiologist's report to the effect that there was great enlargement of the heart.

The *physical signs* relate to the sternal deformity and to the altered shape and position of the heart; they have been summarized in Table I.

The sternal depression. It has been conventional to refer to this deformity of the lower end of the sternum as funnel depression, but when the present series was examined it became evident that the effects on the heart depended directly on the extent of the backward bulge of the sternum. Adopting the same simile, three types may be described, the *funnel*, the *cup*, and the *saucer* depression, according to the depth of the sternal pit (Fig. 1). Thus, the funnel type is deep and usually narrow at the apex (Fig. 2 and 3), and the cup variety is moderately deep and usually rounded at the apex, while the saucer type is a shallower and wider depression. The antero-posterior measurement of the chest in the two cases with funnel depression was $4\,1/2$ and 5 inches respectively; in eight cases of cup deformity it varied from $5\,1/4$ to $5\,3/4$ inches, and in the six cases with saucer depression it was 6 or $6\,1/4$ inches. The same measurement for the control series of 16 healthy subjects was never less than 7 inches and the average was $7\,3/4$ inches (Table I). The lateral measurement was unaffected by the sternal deformity and the average of 10 inches was the same for the control series.

The apex beat. This was displaced to the left in all 16 cases; this effect was obvious in the six instances of saucer depression, but it was greater in the ten cases with either cup or funnel depression (Fig. 4 and 5), and in these it often appeared beyond the anterior axillary line. Although scoliosis was present in nine, it only contributed to the displacement in four and even in these in only a small way. In the two cases showing the severe or funnel form of depression the shift of the apex beat had resulted from the escape of the heart to the left away from the grasp of the jaws formed by the sternal bulge and the spine. In the 14 cases with cup or saucer depression the right border of the heart had maintained its normal relationship to the spine, although sometimes it was lifted a little, so that the outward shift of the left

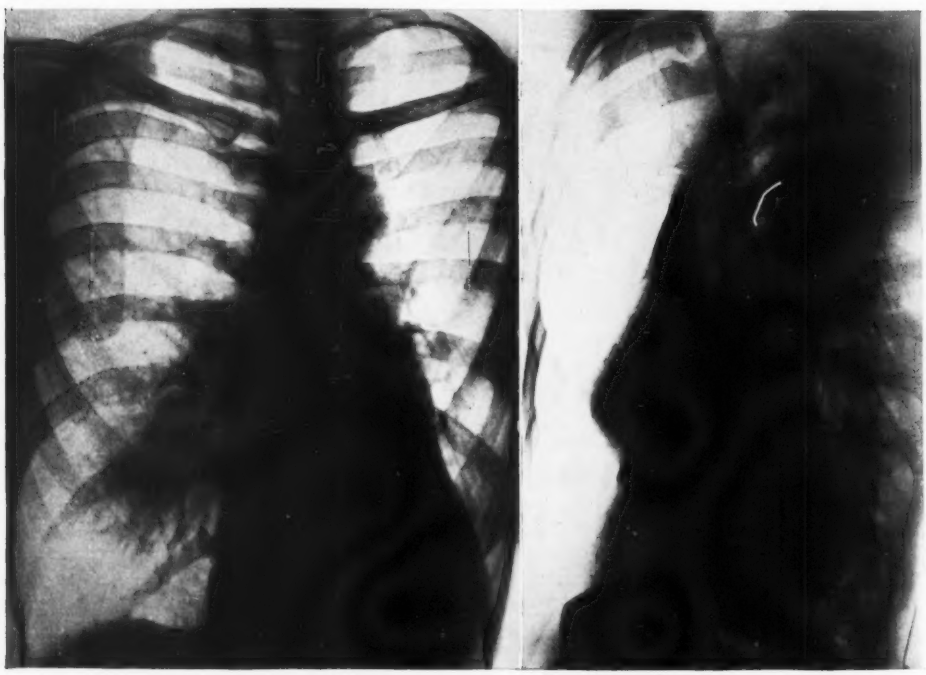

FIG. 4.—Anterior and left (II) oblique teleradiograms in Case 1 with funnel depression. The heart is displaced backwards and to the left.

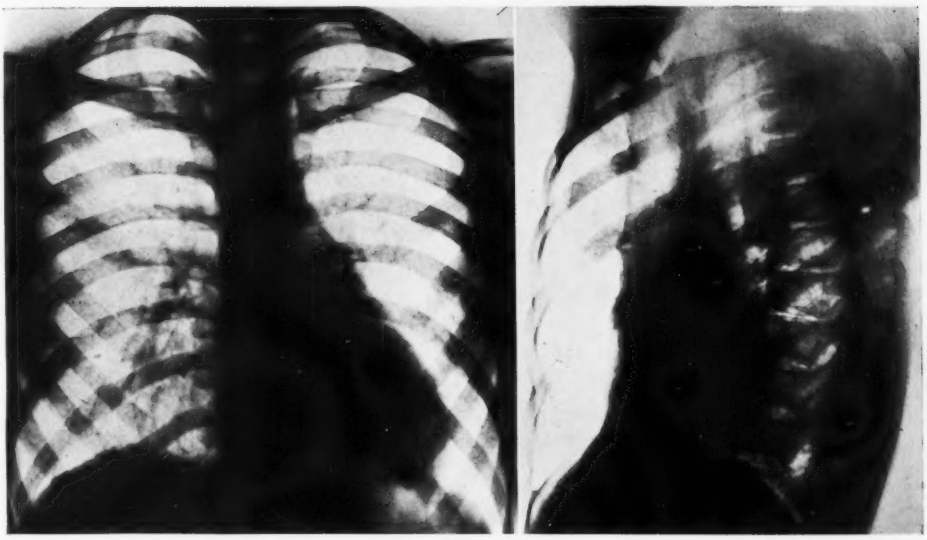

FIG. 5.—Anterior and left oblique teleradiograms in Case 2 with funnel depression. The heart is displaced bodily to the left.

border and the apex beat was the outcome of flattening or compression of the heart within the jaws of the sterno-spinal vice.

Where physical activities had been limited on medical advice, displacement of the apex beat had been accepted as evidence of cardiac enlargement and as one reason for imposing the unjustified restrictions.

The heart murmur. A systolic murmur, heard best at the left edge of the depression, was present in all. It was sometimes moderately loud; in three cases it was short and accompanied by splitting of the first heart sound which formed the chief auscultatory sign. The presence of the murmur had always seemed to add significance to the displaced apex beat and had increased the suspicion that organic heart disease had produced cardiac enlargement. Actually the murmur was of the innocent kind and was situated in mid-systole; it might have been produced by the impact of the heart on the sternal bulge, for the murmur was louder in the cup depression than in the saucer variety.

Radiological findings. The wider application of cardioscopy (radiological examination of the heart) in the investigation of patients suspected of heart disease, has in the meantime tended to confirm rather than remove the invalidism imposed after clinical examination of subjects showing depression of the sternum. This has arisen from a failure to discern that the enlargement of the cardiac silhouette in such cases results from a change in the shape of the heart and not from an alteration in its size.

In the two cases of funnel depression (Fig. 2 and 3) the heart was displaced bodily to the left (Fig. 4 and 5), and without changing the shape or size of the heart shadow in either the anterior or oblique views.

In the eight cases of cup depression (Fig. 6 to 9) there was enlargement of the anterior cardiac silhouette with prominence of the pulmonary arc. The enlargement was moderate in four (Fig. 10 and 11), and slight in the other four (Fig. 12). That such enlargement was apparent and not real was confirmed in the oblique views where the heart shadow was normal in size or sometimes seemed small.

In the six cases of saucer depression (see Fig. 15, p. 169), enlargement of the heart shadow in the anterior view was present but slight (Fig. 13), yet the condition is no less important on that account. Indeed, since some of the other signs of sternal depression are less conspicuous in this variety, the recognition of the effects of saucer depression needs even greater

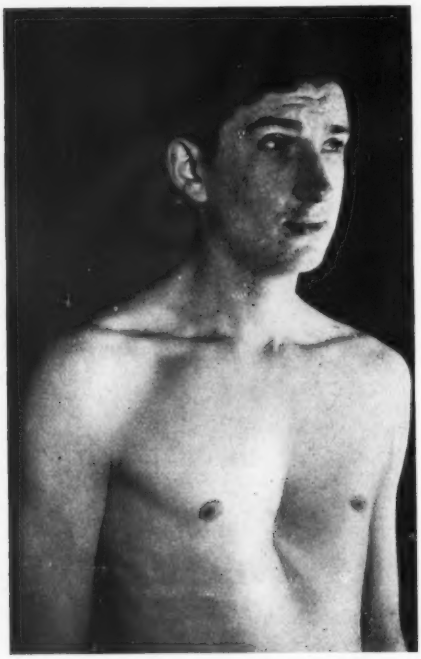

FIG. 6.—Cup depression in Case 4. A–P measurement of 5½ inches against 8 inches in control.

FIG. 7.—Cup depression in Case 5. A–P measurement of 5½ inches against 7½ inches in control.

FIG. 8.—Cup depression in Case 6. A–P measurement of 5½ inches against 8½ inches in control.

FIG. 9.—Cup depression in Case 10. A–P measurement of 5¾ inches against 8½ inches in control.

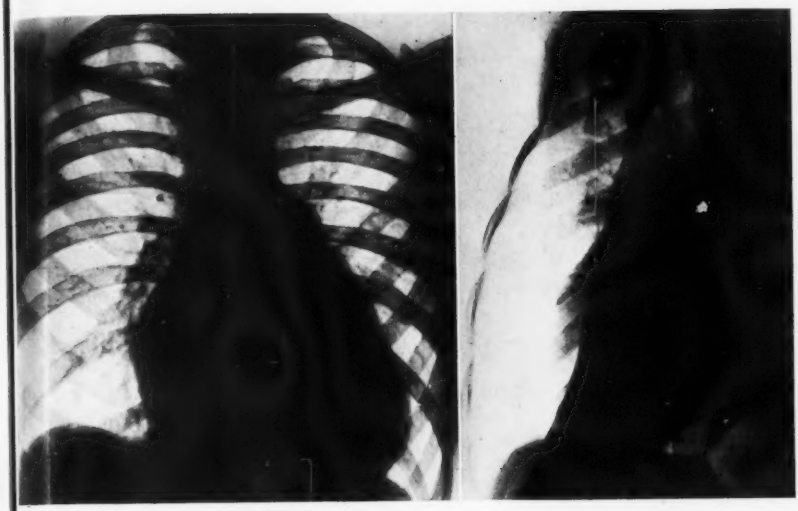

FIG. 10.—Anterior and left oblique teleradiograms in Case 4 with cup depression. The heart shadow is enlarged and the pulmonary arc is prominent in the anterior view, but the heart is normal and moved backwards in the left oblique.

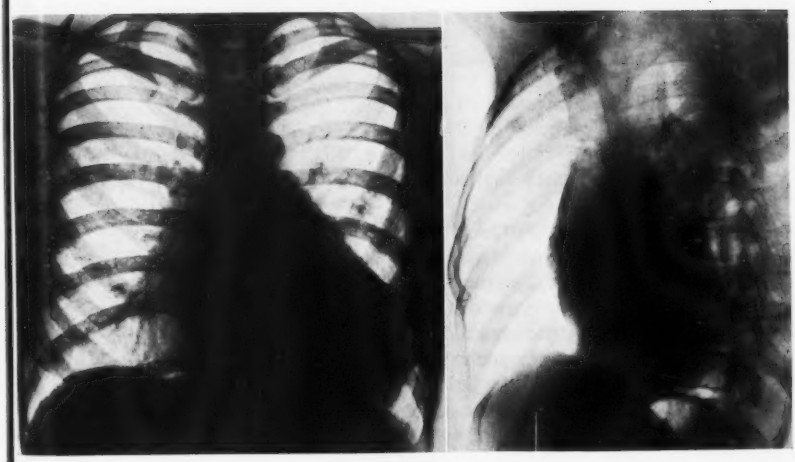

FIG. 11.—Anterior and left oblique teleradiograms in Case 10 with cup depression. The heart shadow is enlarged in the anterior view, but normal and displaced backwards in the left oblique.

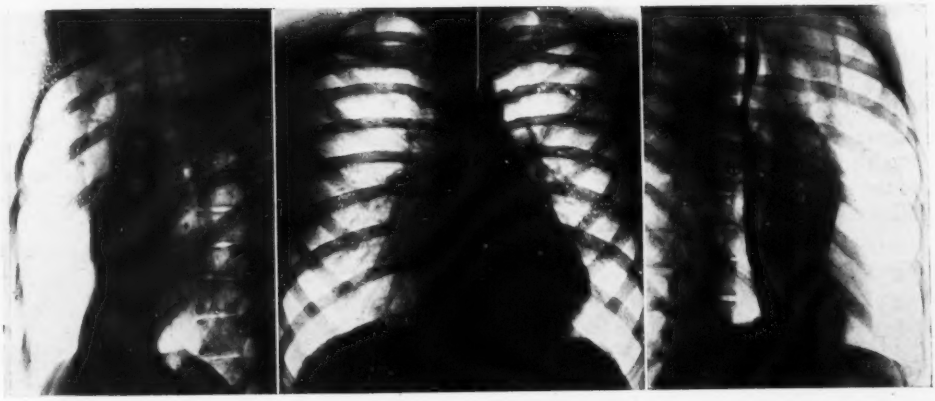

FIG. 12.—Anterior and left and right oblique teleradiograms in Case 5 with cup depression. The heart shadow is moderately enlarged in the anterior view but normal in the obliques.

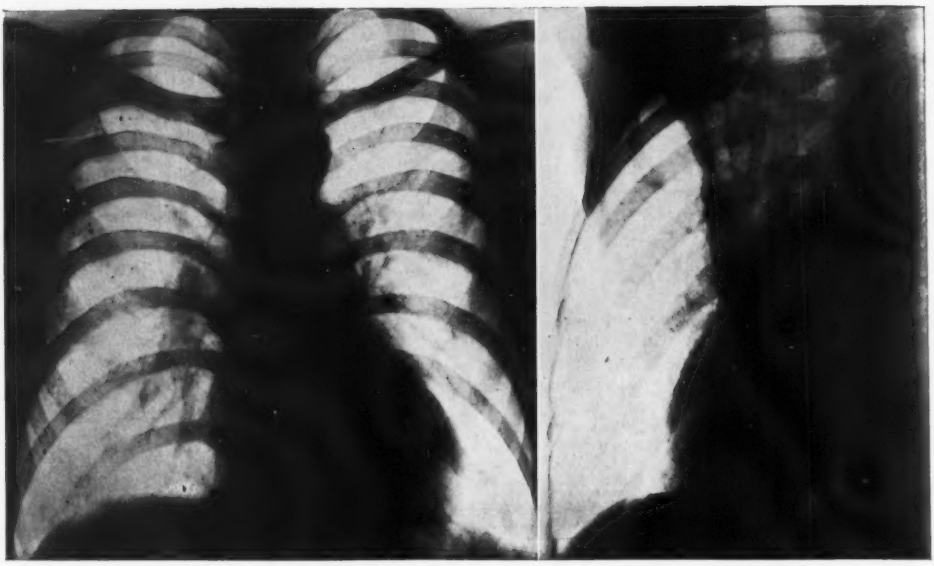

FIG. 13.—Anterior and left oblique teleradiograms in Case 13 with saucer depression. Slight enlargement of the heart shadow in the anterior view, but normal and displaced backwards in the left oblique.

emphasis. As with cup depression so with the saucer variety the heart shadow appeared normal in size in the oblique views.

In addition to the enlargement of the cardiovascular silhouette in the *anterior* view another characteristic feature during cardioscopy was the transparency of the shadow. Thus the spine and ribs were plainly discernible through the heart, and so light was the heart shadow to the right of the sternum that it was sometimes difficult to make out on screening alone the limits of the right border; the right auricle was also often lifted by the sternal depression below causing widening of the right cardiophrenic angle; emphysema was not a factor in the production of such changes. Immediately to the left of the spine a band-like area especially lacked the customary density of the heart shadow in this position. Naturally such transparency was absent in the funnel depression, and was more obvious in the cup depression than in the saucer variety. In the *left* (II) *oblique* position, apart from the size of the heart appearing normal or even smaller, a characteristic finding was its displacement backwards to overlap the adjacent bodies of the vertebræ. Similarly the aortic window (the light area below the aortic arch) was constricted by the approximation of the cardiovascular pedicle to the spine. This trespass on the clear areas provided by the aortic window and the retrocardiac space in this view, caused by the fusion of the cardiovascular and spinal shadows, makes it difficult to identify the separate structures and impoverishes the picture. Such effects were naturally more noticeable in the funnel and cup depression than in the saucer variety. The heart was also normal or narrow in the *right* (I) *oblique* position where the bony deformity in relation to the heart was seen to best advantage. There was no abnormal displacement of the barium-filled œsophagus apart from a slight shift to the left in the two cases with funnel depression.

No significant changes were found in the *electrocardiogram* except that the S wave in lead I was sometimes deep (Fig. 14).

DISCUSSION

Depression of the lower end of the sternum is a common deformity and Lang (1928) found it in 3·5 per cent of children in the first year of schooling. This incidence makes it important to recognize and accept the innocent effects that the deformity may produce on the heart so as to guard against misinterpreting these for the more sinister signs connected

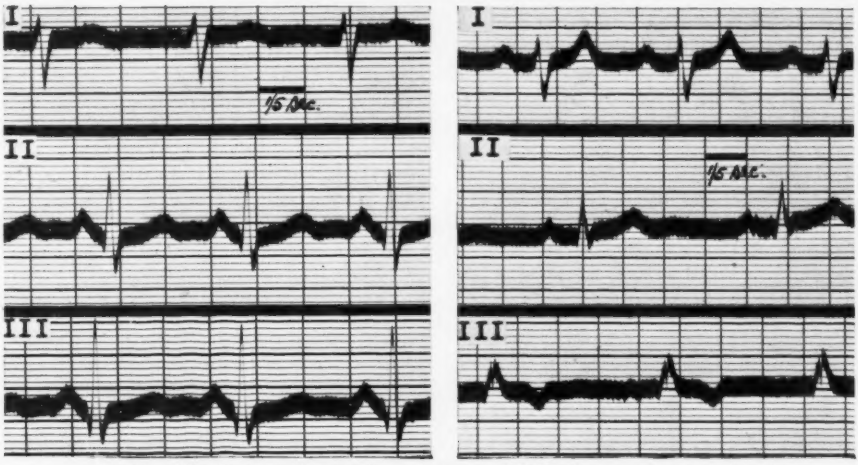

FIG. 14.—The electrocardiogram from Cases 1 and 7 showing deep S wave in lead I.

FIG. 15.—Saucer depression in Case 13. A–P measurement of 6 inches against $7\frac{1}{2}$ inches in control.

with true cardiac enlargement and the conditions that give rise to it. There are many factors said to play a part in the evolution of this sternal deformity, such as abnormal length of the ribs (Flesch, 1873), occupations like cobbling (Laennec, 1819), intra-uterine arrest of the development of the sternum (Ebstein, 1882), and post-natal abnormal development in a child of slender build arising from faulty posture (Kuhns, 1931). Here there was no evidence that any had resulted from congenital maldevelopment, and in all it appeared in early childhood when it might have been the outcome of a disturbance in the mechanism of respiration.

Greatest interest lies with the changes in the heart shadow on cardioscopy. Cardiac hypertrophy was reported by Bein (1912), Frühwald (1913), and Findlay (1921), while Roesler (1943) spoke of circulatory embarrassment. No such effects were observed in my series; the cardiac silhouette was always enlarged in the anterior view in both the cup and saucer depression, but the heart was merely altered in shape and not in size, for it was normal and sometimes small in the oblique views. The explanation of the irregular findings on cardioscopy comes from a study of the thoracic measurements in sternal depression. The antero-posterior measurement of the heart in a series of necropsies on adults was about 3 inches, and the thickness of the sternum, vertebral column, and the soft tissue covering them was about $3\frac{3}{4}$ inches. Thus when the external antero-posterior measurement of the chest is $6\frac{3}{4}$ inches the heart assumes its natural contour and is not subject to external pressure.

In 16 subjects serving as control cases for the present series, being of the same age and of comparable build, the external antero-posterior measurement of the chest was never less than 7 inches and the average was 7 3/4 inches. If this measurement, however, is less than 6 1/2 inches in an adult the approximation of the sternum and spine is such as to compress the heart and reduce its antero-posterior measurement, extending its lateral limits and so enlarging the anterior shadow on cardioscopy.

Should enlargement of the heart fail to be explained by disease, it is necessary to entertain a diagnosis of flattening of the heart by sternal depression, and to assess the size of the heart in the left oblique position, observing at the same time its relation to the spine. Confirmation of the condition is readily obtained from the external antero-posterior measurement of the chest. Such measurement for the series (Table I) was never greater than 6 1/4 inches, so that this figure or a lesser one predicts for the subject a widened cardiac silhouette in the anterior view or displacement in the case of funnel depression. For this reason a measuring calliper is an indispensable instrument whenever an opinion is sought on such a radiological appearance of the heart.

Summary and Conclusions

Sixteen adults in whom there was a moderate or greater degree of depression of the sternum were examined especially for its effect on the heart. The association of a displaced apex beat and a systolic murmur with the deformity had led to a restriction of physical activities in every case; this invalidism had been enforced more rigidly whenever radiological examination had been added, because it had shown a big heart shadow. The symptoms were never once the result of heart enlargement or embarrassment, but were the direct outcome of the physical inactivity or mental anxiety resulting from the unwarranted invalidism.

According to the depth of the depression the cases were separated into three groups. Two cases belonged to the first group of *funnel* depression, where the hollowing was deep and the external antero-posterior chest measurement was 4 1/2 and 5 inches respectively. Eight cases belonged to the second group of *cup* depression, where the receission was moderately deep and its apex more rounded; the external antero-posterior chest measurement in these cases varied from 5 1/4 to 5 3/4 inches. Six cases formed the third group of *saucer* depression, where the dip was shallower and wider and the external antero-posterior chest measurement was 6 or 6 1/4 inches.

The radiological findings were characteristic for each group. Thus, in funnel depression the heart was displaced bodily to the left of the spine and was unchanged in shape or in size. In the cup depression the cardiac silhouette in the anterior view was less dense than normal and was moderately enlarged with prominence of the pulmonary arc; in the left oblique view the heart shadow was normal or small, and was displaced backwards to overlap the spine, thereby obliterating the clear areas of the aortic window and the retrocardiac space. Similar effects, although less in degree, were observed in the saucer depression; the importance of this last group lies in the less obvious chest deformity giving rise to changes which may lead to the erroneous interpretation of the enlarged cardiac area seen on radioscopy if the effects of this saucer depression are not known.

It needs final emphasis that none of the 16 cases with depression of the sternum showed actual enlargement of the heart, nor suffered from symptoms related to the heart. Indeed, they were all healthy and were handicapped only by the restrictions imposed on them by a medical examination that had misinterpreted the clinical and radiological signs.

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